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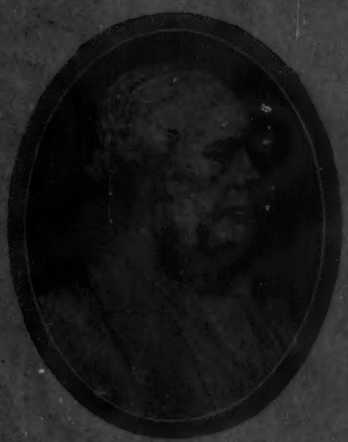

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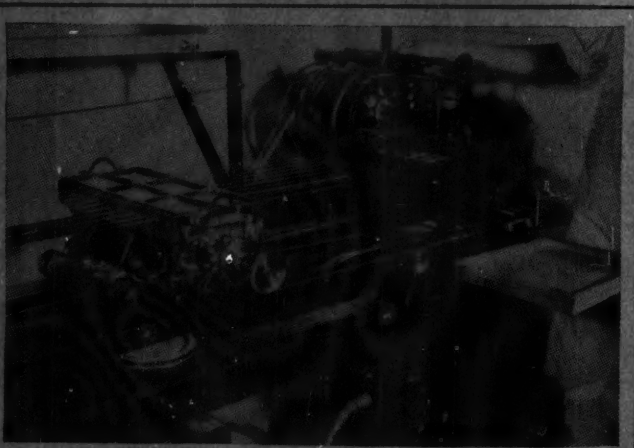

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CAISSON DISEASE (COMPRESSED-AIR ILLNESS) OF BONE WITH A REPORT OF A CASE

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CAISSON disease—compressed-air illness—is a well-known clinical syndrome, but its late manifestations, which rarely occur, are less well recognized.

CASE REPORT

HISTORY.—On April 10, 1941, P. F., aged 37, a general labourer, was seen at the Royal Northern Hospital, complaining of pain in both shoulders and in the right hip. The pain had been intermittent for the last nine months.

There had not been any history of previous injuries, nor of past diseases, such as rheumatism or tuberculosis.

ON EXAMINATION.—The patient appeared to be a healthy man of normal development. His shoulder-joints had a full range of active and passive movements, which were almost painless; bony crepitus could be felt on both sides; this was more marked on the right than on the left side. Movements of the knees and hip-joints were full and painless.

Radiographs of the shoulder-joints showed bony disease in the articular surfaces of both humeri

FIG. 301.—Radiograph of the right shoulder-joint, showing changes in the articular surface of the humeral head.

FIG. 302.—Radiograph of the left shoulder-joint, showing early changes in the articular surface of the humeral head.

(*Figs.* 301, 302). The right knee and both hips appeared to be normal; unfortunately the rest of the skeleton was not examined radiographically.

On being questioned as to whether he had worked in a caisson, he replied that he had been employed in underground work until 16 months ago, and prior to that he had worked in compressed air for four years. During that time he was under medical

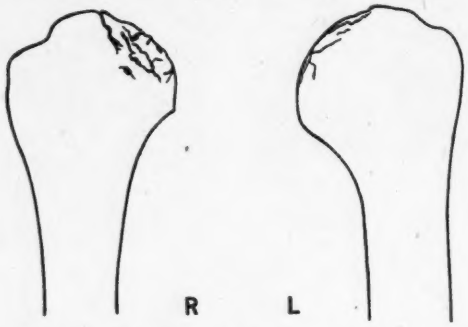

FIG. 303.—A tracing of the films of *Figs.* 301, 302, showing more clearly the extent of the radiographic changes.

supervision and was examined periodically. He had worked mostly with a pick and shovel.

He had had an occasional epistaxis and at times had developed cramplike pains in the limbs—'the bends'; these mild symptoms did not worry him, for he said that they soon passed off; he had never at any time lost consciousness, nor had had any paralytic manifestation of caisson disease.

Since finishing this underground work, 16 months ago, he had been doing similar general labouring with a pick and shovel at ground level, until three weeks previous to his being seen at hospital.

RADIOLOGICAL APPEARANCES.—

1. *The right head of humerus* (*Fig.* 301): This shows a depression in the contour of the middle third of the articular surface; the underlying bone presents a mosaic appearance, the fragments of which appear denser than the surrounding bone, each being separated by irregular thick fissures.

The appearance is that of an avascular necrosis of a portion of the articular surface; the rest of the shoulder skeleton appears to be normal.

2. *The left head of humerus* (*Fig.* 302): This shows an earlier change, but to a less marked degree. The middle third of the articular surface, i.e., a similar site, likewise shows a depression, and thin lines of fissuring can be seen radiating into the underlying bone. Fragmentation is not gross and its density is uniform with the rest of the bone.

SUBSEQUENT HISTORY.—Following his first attendance as an out-patient on April 10, 1941, he attended again the following week, when he said his symptoms were less; he was ordered a short course of heat therapy to the shoulder regions; he did not attend for this treatment, as he resumed work on his own accord a few days later.

After two months, it was learnt by chance that he was dead. He died on June 12; that day he was seized with a severe pain in the chest, and collapsed fatally in the street.

POST-MORTEM EXAMINATION.—Dr. James Davidson found that death was due to coronary thrombosis; he reported that the heart was slightly enlarged, there was some dilatation of all chambers; the myocardium was of average thickness and appearance; "valve cusps normal; tricuspid valve admitted four fingers; mitral admitted three fingers. At commencement of the interventricular branch of the left coronary artery, there was a patch of atheroma with narrowing of the lumen, and here there was a well-formed thrombus formation of recent origin, firmly adherent to the wall".

Examination of the other organs revealed little of note: the lungs were somewhat emphysematous; the alimentary tract, spleen, pancreas, kidneys, suprarenals, and brain were all normal.

Unfortunately, the writer did not see the routine autopsy, for it was only after its conclusion that he was fortunate in obtaining the post-mortem specimens of the upper ends of the humeri (*Figs.* 304, 305). These will be described in some detail.

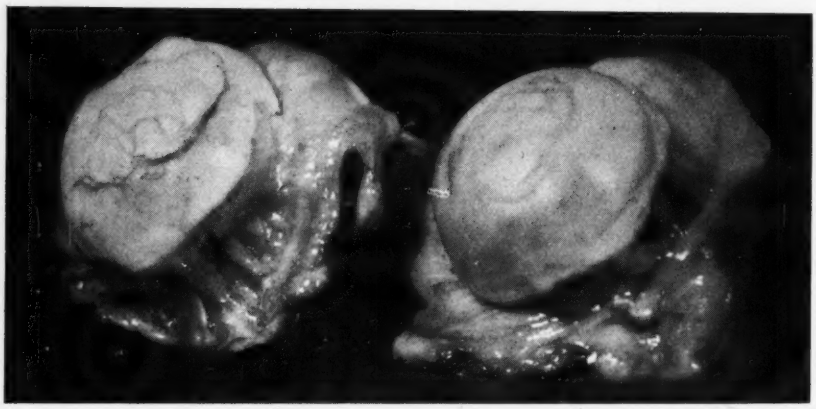

FIG. 304.—A photograph of the post-mortem specimens of both heads of the humeri; the articular surface of the right humerus (on the left side of the photograph) shows a more advanced and extensive change.

PATHOLOGICAL APPEARANCES.—

1. *The right head of humerus* (*Figs.* 304, 305).—

Macroscopical changes: The articular surface shows an irregular area of devitalized cartilage, it is paler than the surrounding normal articular surface. This area covers about two-thirds of the head of the humerus; it presents a mosaic appearance, with irregular fissures which divide its surface into small white calcified areas; at its upper edge it is in the process of separating from the underlying living bone;

here it can be sprung in and out by the application and release of pressure; it represents an area of avascular necrosis.

There is evidence of some osteophytic formation at the edges of the articular cartilage. The capsule appear to be paler and denser than the smooth shiny cartilage around. There is no evidence of any separation; the disease appears to be less extensive and of later development than that on the right side.

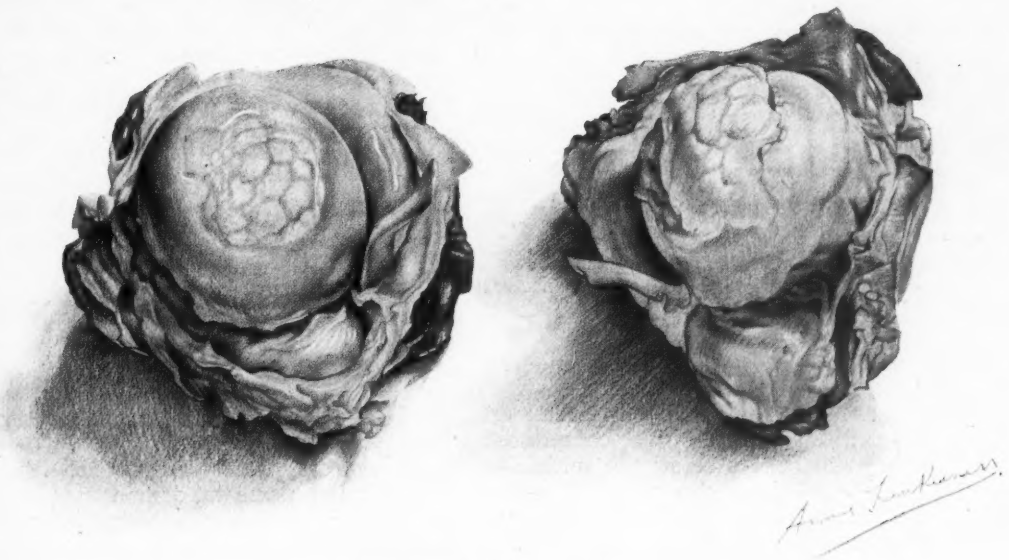

FIG. 305.—Drawing of the specimens.

appears normal, except for small areas of hyperæmia on its velvety surface; the synovial fluid was not in excess. The glenoid surface of the shoulder-joint was also normal.

Microscopical examination of the capsule only was made; this was of normal appearance. (A section of the diseased portion of bone was taken from the *left* side only; this was examined microscopically, *see below*.)

A wedge-shaped section through the diameter of the diseased area was cut and removed; it revealed that the infarct extended to about a ½ in. below the articular surface of the humerus; the line of demarcation was obvious, for the dead bone, which was paler, contrasted against the darker cancellous bone below; this line of contrast extended gradually to the surface, reaching it at the edge of the necrotic area.

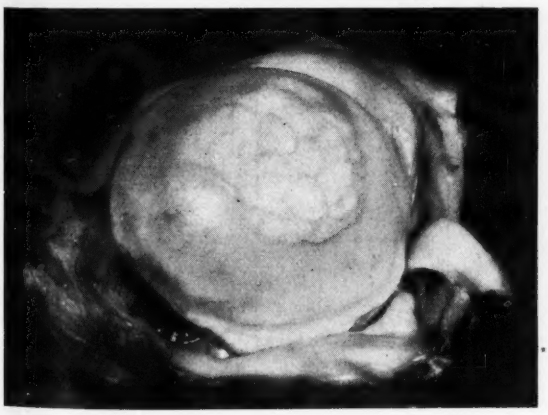

FIG. 306.—Another view of the articular surface of the upper end of the left humerus; the fissured appearance of the area of avascular necrosis can be seen.

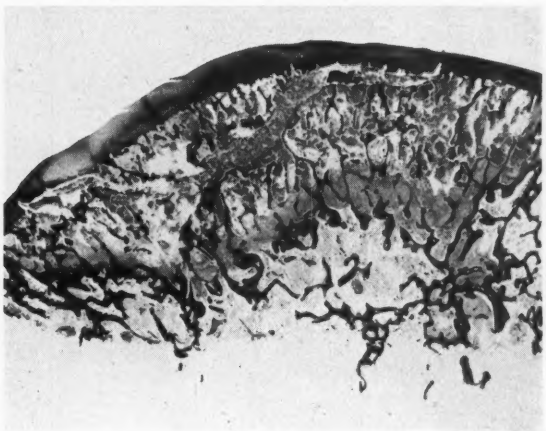

FIG. 307.—A low-power view (× 2·4) of a section through the upper articular surface of the left humerus; the depth of the aseptic necrosis can be seen.

2. *The left head of humerus* (*Figs.* 304–306).—*Macroscopical changes*: This articular surface also shows an irregular depressed area of devitalized cartilage, covering about one-third of the head of the humerus. This portion is covered with ridges, which divide the surface into small irregular areas; these

There is a little osteophytic lipping of the bone at the margin of the articular surface; the capsule appears normal.

Microscopical changes (*Fig.* 307): The section was examined by Dr. R. W. Scarff, whose report is as follows: "The section consists of a portion of

cancellous bone covered on one surface by articular cartilage, the whole measuring 38 × 18 mm. Immediately under the cartilage is an area of necrosis extending for 35 mm. beneath the cartilage and 10 mm. in depth. The dead bone extends up to the cartilage, which appears healthy, but has a zone of calcification on its deep surface. There is a suggestion of healing of a small superficial erosion.

"There is persistence of structure in the centre of the lesion with complete absence of nuclear staining and no sign of osteoclastic activity.

"On the deep surface of the area is a layer of fibrosis which also shows focal calcification. This layer shows new-bone formation; in places new bone is deposited on the trabeculæ of dead bone. Also at the junction are seen areas of reaction to fat necrosis—histiocytes, inflammatory cells, etc.

"The appearances are those of an aseptic necrosis of bone and are compatible with a sudden vascular occlusion some time previously."

DISCUSSION

Caisson disease is well recognized in civil life; its symptoms are liable to develop in divers and those who work in caissons in a compressed atmosphere at great depths below the surface of the earth. The disease is the result of injury to tissues by liberated bubbles of nitrogen, when the body, which in compressed air has absorbed an excess of nitrogen, is too rapidly decompressed.

SYMPTOMATOLOGY

The disease can present various signs and symptoms; the principal being the 'bends', which are severe pains in the joints and loss of power in the limbs; theoretically the 'bends' can affect any part of the body, but they are commonest in the region of large joints.

Dermal changes with pruritus and hæmorrhages may occur.

Dyspnœa, due either to pulmonary or cardiac lesions; paralysis, with spinal cord involvement; and cerebral or alimentary symptoms may also arise. Aural sensations of tinnitus, vertigo, and deafness are also frequent.

These are all early manifestations of compressed-air illness, and are well known; late complications producing changes in the bones and joints more rarely occur.

Skeletal Changes.—The essential lesion is an infarction of bone; the case reported above illustrates the condition occurring in the heads of both humeri.

Recently Kahlstrom, Burton, and Phemister (1939) conjointly described 4 cases with extensive changes in the skeleton; these were revealed in the shafts and the ends of certain bones; many involved the articular surfaces of the large joints; all the lesions appeared to be the result of massive aseptic necrosis of bone. They were of long standing; one was demonstrated post mortem and another by biopsy. Their report gives a good review of the literature and of previously reported cases; they present a full clinical, radiological, and pathological picture of this disease.

Coley and Moore (1940) also have reported 2 cases, both exhibiting multiple infarcts in some of the long bones.

The following table shows the salient features of these recently reported cases; the division of

CAISSON DISEASE. DETAILS OF RECENTLY REPORTED CASES

	Age	Onset and Duration	Articular Site	Non-articular	Remarks
KAHLSTROM, BURTON and PHEMISTER (4 Cases)					
Case 1 ..	61	Attack of caisson disease, 35 years ago	Both hips (Rt., loose bodies) Lt. shoulder (loose body)	Both femora (extensive); Lt. tibia (small); Lt. humerus	DIED of carcinoma of lung; also arteriosclerosis; gallstones; duodenal ulcer
Case 2 ..	37	History of an attack of caisson disease, 4 years ago	Both hips; both shoulders	Nil	Biopsy—Lt. hip
Case 3 ..	54	History of a rapid decompression, 30 years ago	Nil	Both femora; Fibulæ (both upper ends); Tibiæ (upper and lower)	Spastic palsy; also history of syphilis
Case 4 ..	55	Definite history of onset, 21 years ago	Both hips; both shoulders	Both femora (lower ends); Tibiæ (both ends)	
COLEY and MOORE (2 Cases)					
Case 1 ..	57	24 years ago	Rt. hip; Rt. shoulder	Both femora (lower ends); Rt. tibia (upper end); Rt. femur (upper end); Lt. humerus (upper end)	DIED, of cancer of tongue 16 years a caisson worker
Case 2 ..	49	8 years ago, a mild attack	Rt. shoulder	Rt. humerus; Lt. femur (both ends); Both tibiæ (upper and lower ends); Both ischii	DIED, cancer of bronchus 4 months a caisson worker

the lesions into ' articular ' and ' non-articular ' is purely arbitrary, for their pathology and production are similar ; whether the joint surface is involved determines the classification of these infarcts.

In this disease infarcts are found in the long bones ; they may be single or multiple, varying in size, affecting many or few bones. They occur at any site in the femora, humeri, tibiæ, and fibulæ. It is convenient to divide them into articular and non-articular lesions.

The Non-articular Lesions.—These occur in the diaphyses of long bones ; they are usually 'silent', causing no symptoms. Radiologically, these medullary infarcts appear as central areas of dead bone with an irregular thickened outline, which is due to partial replacement by new bone at the margins of the infarcted area. In most cases these areas have been examined a long time after the initial onset. Creeping invasion and substitution of the dead bone by new may have reduced materially the size of the necrotic area before the process comes to a standstill, with calcification of the surrounding fibrous zone. Small areas may become completely calcified. Pathological fractures do not occur.

The Articular Lesions.—These occur in the bone-ends, and commonly affect the hip and shoulder-joints (this case illustrates the latter) ; the articular lesions are commonly bilateral.

An area of aseptic necrosis develops in the cancellous portion of the bone and devitalizes the overlying articular cartilage ; subsequent changes develop. These are : first, a relative increase of density, fragmentation, followed by creeping bone substitution or fibrosis ; these changes are similar to those occurring in ordinary aseptic necrosis elsewhere, e.g., in the head of the femur following a transcervical fracture, or in the proximal fragment of a fractured scaphoid in the hand.

With weight-bearing and continued work, the articular surface collapses, with the gradual development of an arthritis deformans ; loose bodies of osteo-cartilaginous structure may form in the joints.

Symptoms of muscle and joint pains usually appear shortly after a rapid decompression ; these may be accompanied by other manifestations of compressed-air illness. The joint pains are severe, of a boring character, and occur most markedly in the region subsequently affected ; they may last a few hours or a few days, depending on the severity of the attack.

Later, usually years afterwards, if an articular lesion develops, an associated arthralgia will accompany it, which will increase in severity with the development of the inevitable arthritis ; deformities then follow. In most cases the arthritic symptoms occur many years after the initial lesion ; in the majority a previous history of an attack of caisson disease is elicited.

AETIOLOGY

The production of these infarcts is due to bubbles of nitrogen collecting in or around the nutrient blood-vessels, causing an interference with the nutrition of a portion of the bone. An area of avascular necrosis develops.

In compressed-air illness there is a liberation of bubbles of nitrogen from the blood into the tissues ; " respiration under pressure leads to a solution of nitrogen in the blood and tissues ; oxygen and carbon dioxide are also dissolved, but except at very high and prolonged pressure, the body can deal with them and this does not cause symptoms. The degree of solution depends on the pressure and length of the exposure, while the rate depends on the vascularity of the part. A rapid release of pressure results in the effervescence of nitrogen ".

That gas is present in these tissues is proved radiologically ; Gordon and Heacock (1940) report a case of an injured worker in a compressed atmosphere being brought too rapidly to the surface. An immediate radiograph showed a fracture of one tibia into the knee-joint and a localized volume of gas in the synovial sacs of the joint. Radiologically this gas had resolved in six weeks. In this case, it is probable that a number of bubbles of nitrogen were liberated into the joint from along the line of fracture of the articular surface.

Fats dissolve more than five times as much nitrogen as an equal volume of water or blood-plasma (Vernon, 1907) ; this fact probably accounts for the long bones being selected as the site of nitrogen emboli, for these bones are rich in marrow fat.

The presence of end arteries and the relative slowness of the circulation in the bone-marrow also favour the accumulation of nitrogen therein.

Whether the necrosis is due to intravascular or extravascular pressure by nitrogen bubbles is uncertain. Phemister (1939) attempted to produce bone necrosis in dogs by injecting varying amounts of air into their femoral arteries ; although in some he produced partial or complete paralysis of the hind limbs by air emboli, bone necrosis did not develop. He concluded that " the bone lesions in caisson disease are due rather to gas liberated in the medullary cavity under sufficient pressure to asphyxiate the tissues than to emboli ".

In most cases the infarcts, whether in the diaphyses or epiphyses, are multiple and often symmetrical. It is doubtful whether they are all produced by a single attack of caisson disease ; it is possible that after an initial lesion, others may arise later.

Trauma determines the site in some cases ; repeated injuries, as occurring in heavy labour or weight-bearing, add to the destruction of the articular surfaces of the limb bones, if the underlying bone is rendered avascular by infarction.

Aseptic necrosis of bone of undetermined aetiology may occur, though this is rare. Kahlstrom, Burton, and Phemister (1939) cite 4 cases occurring in patients who give no history of having worked in compressed air. They state that no similar records can be found in the

literature of massive multiple infarcts of the shafts and epiphyses of bones except in patients who had worked in a compressed atmosphere. These authors fully discuss the possible causes of the vascular blockage, and conclude that in 1 case the cause was unknown, and in 2 others the possibility of undiagnosed fat embolism was considered, as there was a previous history of bone fractures in the past. If this last assumption was correct, the incidence of bone infarcts so produced would be relatively frequent, as fat embolisms following bony or soft tissue trauma are becoming more frequently recognized.

The exact mode of production of bone infarcts is unknown, for they have not been produced experimentally, this precluding any demonstrable evidence of its initial pathology; it is only in later years that their presence is detected.

TREATMENT

Briefly, the treatment of caisson disease is largely prophylactic; this treatment of slowly regulated decompression and recompression is of greatest value; these measures, which are conducted under strict supervision, prevent its immediate effects.

In recent years, much experimental work has been done to lessen the incidence of nitrogen emboli; the inhalation of helium-oxygen mixtures has been used to eliminate these risks, for helium is much less soluble in the blood than nitrogen, and, being inert, does not affect the body tissues.

In established cases, showing the bony changes as described above, the treatment is on general orthopædic lines. With the articular lesions, the prevention of weight-bearing and heavy work is necessary to lessen any further joint destruction. Areas of dead bone and loose detached fragments should be removed if interfering with joint function.

The non-articular lesions, i.e., infarcts involving the shafts, do not require treatment; for they do not usually cause symptoms; they thus often escape notice. Spontaneous fractures do not occur, so there is little risk with weight-bearing.

CONDITIONS ALLIED TO CAISSON DISEASE IN MODERN WARFARE

Caisson disease is well recognized in civilian workers—the deep-sea diver (Fraser, 1940), and those who work in compressed atmospheres at great depths below the earth's surface are liable to its effects. In some branches of modern warfare there is a similar risk—those engaged in submarine work and high-altitude flying are exposed to these hazards.

In the Navy, submarine crews and deep salvage workers are liable to develop symptoms of compressed-air illness, especially those who ascend from submerged vessels over 300 ft. below the surface of the sea (Jenkinson, 1940).

In the Air Force, the high-altitude aviator, while climbing to the substratosphere, is prone to develop aero-embolism (Armstrong and Heim, 1940), the symptoms of which are identical with those of compressed-air illness (caisson disease). "During ascent in aircraft, or in any other situation in which the atmospheric pressure is decreased, the internal partial pressure of the body nitrogen is above that of the nitrogen in the lungs, and the tissues are therefore supersaturated. As a consequence, the nitrogen dissolved in the blood begins to be liberated in the lungs and the nitrogen in the tissues begins to enter the blood-stream, and by this dual process the body tends to rid itself of its excess nitrogen". Bubbles in the blood and body tissues generally have not been found below 30,000 ft. altitude.

Thus in modern warfare it is possible that the incidence of bone infarcts, produced by nitrogen emboli, may increase; the lesions, though rare, may become evident in later years, but it is hoped that the prophylactic measures which are being perfected may entirely eliminate the incidence of compressed-air illness and its sequelæ.

CONCLUSION

Areas of aseptic necrosis of bone in the upper ends of both humeri are described; these occurred in a labourer who had worked in a compressed atmosphere some months previously. The essential lesions in bone are infarcts, which are produced by nitrogen emboli; these areas are situated in the long bones and are usually multiple.

The disease is of topical interest in time of war as the incidence of compressed-air illness may increase; thus its rare manifestations may arise later in the bones of those who work in the bowels of the earth, soar to great heights, or descend into the depths of the sea.

I am much indebted to Mr. Eric Lloyd for his interest in this case and for suggesting its publication; I wish to thank Miss K. C. Clark, of Ilford Limited, and Miss A. Hathway, of the Royal Northern Hospital, for preparation of all the radiographs and photographs; also Dr. R. W. Scarff, of the Bland-Sutton Institute, Middlesex Hospital, for examining the microscopical section, which was prepared at the Institute; and Miss Anna Zinkeisen for executing the coloured drawing of the specimens.

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TUMOURS OF THE URETER

By THOMAS MOORE, Major, R.A.M.C.
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Modern methods of investigation have brought to light many cases of ureteric tumour which would previously have escaped recognition. The condition is still apt to be missed, however, unless attention is paid to every suggestive symptom and meticulous examination carried out until the cause has been found. When it is remembered that a tumour no larger than a bean has caused death by hæmorrhage (Huffman, 1939), it is realized that no detailed investigation which the urologist may employ can be considered pedantic. In this paper the opportunity is being taken of reviewing the lesion and reporting two further cases.

HISTORY

Both benign and malignant neoplasms occur, but the latter are much the commoner. The first case, reported by Rayer in 1841, although lacking histological confirmation, was undoubtedly a carcinoma. Wising and Blix (1878) were the first to report a case of carcinoma complete with histological confirmation. It was not until 1884 that Davy reported the first case in the English literature. In 1922 Aschner was able to collect 47 cases of epithelial tumours of the ureter from the literature. Scott in 1934 collected 61 cases of primary carcinoma of the ureter, including his own 2 cases. In the same year Lazarus reported 3 cases, and made an extensive review of the literature. Two years later Schillings and Sondervorst were able to collect 113 cases. Writing in 1938, Higgins would only accept 86 of the published cases as being definite primary carcinomata of the ureter. He added 5 cases of his own. Reporting a case in the same year, Rusche and Bacon stated that 96 cases had previously been reported. Foord and Ferrier (1939) reported 6 further cases and collected 139 from the literature. With the cases reported by Foley (1940 and 1939), Jewett (1940), Jolly (1939, 2 cases), Columbano (1938), Cabot (1939, 2 cases), Stang and Hertzog (1941, 4 cases), it can be safely assumed that up to date about 150 cases of primary carcinoma of the ureter have been reported.

Benign tumours are much less common. Lebert (1861) reported the first case, and others have been reported at intervals since. Of the 47 cases of epithelial ureteric tumours collected by Aschner in 1922, 21 were simple papillomata. Melicow and Finlay (1932) were able to collect 28 cases and reported 1 case of their own. A case was also reported by Walters in the following year. After cases were reported by Hunner (1938) and Ramsey (1939), Rusche and Bacon (1940) reported 1 case and were able to find 40 cases in the literature up to that time.

PATHOLOGY

Tumours of the ureter may be either of epithelial or connective-tissue origin.

Epithelial Tumours.—These may be simple or malignant, and the latter may be primary or secondary.

Simple Tumours.—The common simple epithelial tumour of the urinary tract is the papilloma. There is still a great difference of opinion on the classification of all epithelial tumours of the urinary tract. None are really absolutely simple, as the most innocent papilloma has the power of forming implantation deposits and there is a gradual transition in type from it to the most malignant carcinoma. Acknowledging this to be true, in this country a simple papilloma is a well-defined condition. It is a neoplasm consisting of a central core of fibrous tissue and blood-vessels, which has many terminal branches, each of which is covered by transitional epithelium. There is no infiltration of the surrounding structures. Such a neoplasm commonly occurs in the bladder, but an exactly similar one may be found in the ureter. It is usually single and in the lower third, but may be associated with other deposits in the ureter, bladder, or renal pelvis, when it is often impossible to say which is the primary growth. Probably they all arise independently. Several cases of simple ureteric neoplasm reported have been presented as polypi. There is some confusion between the two conditions as they occur in the urinary tract; the latter is a solid simple swelling consisting of fibrous tissue and blood-vessels, and covered by transitional epithelium. It is usually associated with chronic irritation and is of inflammatory origin. However, a true papilloma may become œdematous and solid looking due to constriction of its pedicle and simulate a polypus very closely (*see Case* 1). Careful examination reveals its true nature.

Malignant Tumours.—Ureteric carcinomata, like their counterpart in the bladder, may be divided into papillary and non-papillary types. In each the important point is that infiltration of the ureteric wall occurs. Very rarely some gland formations can be seen microscopically so that an adenocarcinoma can be separated. Of the cases reported by Foord and Ferrier, 75 were

papillary, 65 solid non-papillary, and 3 adenocarcinoma. Carcinomata are commonest in the lower third of the ureter, although any part may be affected. They may occur at any age, but are commonest in the later decades of life. The sexes are equally affected. The neoplasm may spread round the entire lumen of the ureter or affect only part of the wall. Direct infiltration of the ureteric wall also occurs, and surrounding structures may be involved. Spread may also occur to the regional lymph-nodes and by the blood-stream to any part of the body, particularly the liver and the lungs. A case has even been reported where the brain was explored for a tumour which proved to be a metastasis, from a primary neoplasm of the ureter (Hunt, 1927).

Secondary malignant epithelial tumours: The ureter may be involved by direct spread from neoplasms of surrounding structures. In the case of the uterine cervix this is not uncommon. Metastatic deposits spread by the blood- or lymph-streams are much rarer. Lazarus (1941) has reported a case and was only able to collect 17 cases from literature. The sites of the primary neoplasms were: prostate, 7 cases; bladder, 3 cases; cervix, 2 cases; urethra, 1 case; stomach, 1 case; bronchus, 1 case; breast, 2 cases. He remarks on the absence of any case of metastatic ureteral deposit derived from a neoplasm of the kidney. However, examples have been reported following hypernephroma by Thomson-Walker (1920), Schacht (1931), and Alstein (1935). A case of a metastatic deposit of the ureter derived from a carcinoma of the kidney is reported in this paper. Somner (1932) reported a remarkable case of a metastatic deposit of carcinoma in one ureter secondary to a primary neoplasm of the other. It is, of course, not very uncommon to get secondary deposits of papilloma in the ureter secondary to a malignant papilloma of the renal pelvis. The underlying pathology of these cases is not fully understood. The deposits arise either as surface implantations from the primary neoplasm or all arise independently due to some underlying aetiological agent circulating in the blood. The ureter may be the site occasionally of secondary malignant deposits which have been spread by transcœlomic migration from some primary intraperitoneal carcinoma.

Non-epithelial Tumours.—These are much less common. They may arise from any of the non-epithelial structures of the ureteric wall; thus myomata, fibromata, and myxomata, etc., occur. Occasional cases have been reported both of the simple and malignant types of non-epithelial tumours. Ravisch (1935) reported an interesting case of neurofibroma of the ureter. Bergendahl (1934) reported a case of sarcoma of the ureter. Spread may occur from this lesion in exactly the same way as in the carcinoma. The condition is so rare that it does not warrant further discussion.

CONSEQUENT PATHOLOGICAL CHANGES

1. Ureteric Obstruction.—Any neoplasm of the ureter, whether simple or malignant, may lead to obstruction with consequent hydronephrotic changes. In one case these were so extreme that rupture occurred (Ramsey, 1939). These obstructed ureters are very predisposed to secondary infection, with all its dire possibilities.

2. Fistula Formation.—Malignant neoplasms in their extension invade surrounding structures, so that a uretero-intestinal or ureterovaginal fistula may be formed.

3. Mechanical Results.—An abnormal structure in a muscular tube leads to increased peristaltic contractions of that tube in an effort to expel the intruder. The ureter thus attempts to expel a papilloma. This leads to elongation and pedunculation of the papilloma, so that it may prolapse through the ureteric orifice. In some cases this only happens when a peristaltic wave passes down the ureter and the neoplasm can be seen popping in and out of the ureteric orifice at cystoscopy. Intussusception of the ureter may also occur, as in the case reported by Hunner (1938) and probably in *Case* 1. The action of the ureter in trying to expel the papilloma leads to constriction of its base, so that it may become œdematous and solid looking, and to the naked eye may simulate a polyp.

CONCOMITANT LESIONS

Cases of ureteric neoplasms have been reported in association with all the common congenital abnormalities of the urinary tract. One case of neoplasm in a ureteric diverticulum has also been reported. In some cases the neoplasm has been associated with calculi. Of the cases reviewed by Lazarus 14·7 per cent were associated with calculi.

AETIOLOGY

The exact cause of ureteric neoplasms is no more understood than that of those occurring in other parts of the body. However, one or two facts do stand out. In a definite proportion of cases they are associated with calculi, and it is possible that the chronic irritation so caused has initiated the neoplastic changes. It is more probable that the association is incidental or that the calculi are secondary to urinary stasis caused by the neoplasm. If calculus were a potent factor in the causation ureteric neoplasms would be very much more common. There is no doubt that certain dyes of the aniline series are closely related to the onset of bladder neoplasms in people employed in this industry and the same is true of ureteric neoplasms, although they are very much less common. Müller (1940) reported a case of ureteric neoplasm which was apparently caused by benzedrine.

SYMPTOMATOLOGY

The commonest symptoms are hæmaturia, pain, and tumour. *Hæmaturia* occurred in 70 per cent of the cases reviewed by Foord and Ferrier. It is unfortunate that it is usually such a late symptom, as it is the one which is most likely to bring a patient for medical advice. It may or may not be associated with pain.

Pain occurs in about 60 per cent of the cases, and may be one of three types: (*a*) *Ureteric colic*, caused by the ureteric obstruction or blood-clots being passed down the ureter. (*b*) *Renal ache*, due to ureteric and pelvic distension. (*c*) *Referred pain*, due to involvement of surrounding structures.

Tumour: This is either a large obstructed kidney or the neoplasm itself. In quite a percentage of cases a swelling is the first symptom observed by the patient.

In late cases loss of weight is noticed and where infection has occurred the usual general signs are present.

DIAGNOSIS

On clinical examination the enlarged kidney or rarely the neoplasm itself may be felt. Occasionally a careful rectal or vaginal examination may be rewarded by feeling the neoplasm. It is well to remember they are more common in the lower third of the ureter. Examination of the urine must never be omitted. When the hæmaturia is macroscopic typical ureteric clots may be found. In other cases red blood-cells will only be seen under the microscope. Very rarely tumour cells may be demonstrated by microscopical examination of the urine.

Radiological examination is always necessary. This is important in excluding opaque calculi and may show an enlarged kidney shadow. Intravenous urography in late cases shows a loss of function of the kidney on the affected side, but in early cases may show a dilated ureter and perhaps a filling defect. Cystoscopy and retrograde urography are the most valuable diagnostic methods. Inspection of the ureteric orifice is very important. Blood may be seen issuing from the affected side. If the neoplasm is low in the ureter the blood forms a constant dribble, while if it is high in the ureter it spurts out with each peristaltic wave in the usual way. In a good proportion of cases the tumour has prolapsed through the ureteric orifice and can be easily seen. In other cases it is only seen as each peristaltic wave forces it out. In some cases the only abnormality seen is a slight congestion of the orifice on the affected side, while in others it may be quite normal. If a definite diagnosis has not been made by inspection alone a ureteric catheter must be passed up the suspected side. In many cases it is obstructed at the site of the tumour, and the retrograde ureterogram will demonstrate this. Sometimes the catheter passes the tumour, but leads to bleeding—a sign first described by Chevassu and Mock in 1912. Occasionally blood can be seen issuing from the ureteric orifice or draining down the catheter, but once the catheter is advanced past the tumour to the renal pelvis clear urine is obtained. If the tumour is low in the ureter bleeding may be seen to be provoked by vaginal examination. Careful retrograde urography is of the highest importance, as apart from seeing the prolapsed tumour a filling defect in the ureterogram is the most valuable sign of the lesion. Rusche and Bacon suggested that the catheter should be withdrawn a few centimetres at a time and the dye injected after each withdrawal, so that a good film might be obtained of the diseased part of the ureter. It has always been my practice in performing retrograde urography to fill the renal pelvis with the medium and obtain a film. The catheter is then withdrawn a calculated distance so that its tip is only about one inch up the ureter. Further dye is then injected and an excellent ureterogram obtained. In this way no abnormality of the ureter is missed. I have not found it necessary to use a Garceau catheter or one with a special bulb near the tip in order to stop backflow of the medium alongside the catheter in order to get a good ureterogram. It is also advisable to take the films in series as advised by T. D. Moore to make sure that any filling effect is really constant.

The most important findings for diagnostic purposes are unilateral hæmaturia, ureteric obstruction, or filling defect. Non-opaque ureteric calculus can lead to error, but the ureterogram in such cases is fairly characteristic. Albarran in 1906 made the first correct pre-operative diagnosis, the tumour being seen through the cystoscope. The following table shows the pre-operative diagnosis in the cases reviewed by Foord and Ferrier.

Tumour of ureter	54
No diagnosis	34
Hydronephrosis	7
Tumour of kidney or ureter	6
Tumour of kidney	6
Stone in ureter	5
Tumour of bladder	4
Tumour or stone in ureter	3
Tumour of gastro-intestinal tract	2
Retrovesical tumour (? origin)	2
Tumour of pelvic organs	2
Ectopic kidney	1
Pyonephrosis	1
Ureterocele	1
Prostatic hypertrophy	1
Prostatic abscess	1
Rectal cancer	1
Dermoid cyst	1
Osteosarcoma of ilium	1
Tumour of ovary	1
Salpingitis	1
Carcinoma of undetermined origin	1
Stricture of ureter	1
Tuberculous ureter	1
Intestinal or ureteric tumour	1

It has not uncommonly been reported that nephrectomy has been performed for unilateral hæmaturia and yet the cause has not been found in the kidney. When the bleeding has recurred the remaining portion of the ureter has been removed and found to contain a neoplasm.

PROGNOSIS

In simple papilloma, if adequate care is taken not to infect other tissues at operation, the prognosis is good. The outlook in carcinoma, however, is very bad. The growth is usually very advanced before the diagnosis is made and there is a considerable immediate operative mortality. Of the 44 cases collected by Foord and Ferrier which had been treated by one-stage uretero-nephrectomy, there was an immediate mortality of 40 per cent; in 22 treated by two-stage uretero-nephrectomy the immediate mortality was 5 per cent. In 100 cases collected by the same authors there was 34 per cent mortality at the end of three months. Scott could only find 2 cases reported in the literature up to 1934 who were alive five years after operation. Higgins reports that all his 5 cases died within two years of operation. Very few cases are alive five years after radical treatment. One case reported by Krafft was alive eleven years after operation. Another reported by Crance and Knickerbocker was alive eight years after operation. Vorpohl reported a case which died from general metastases eight years after operation.

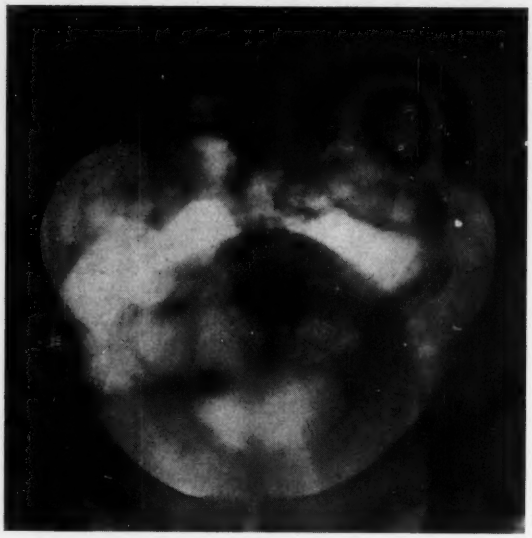

FIG. 308.—*Case* 1. Intravenous urogram showing dilatation and irregular filling defect of the lower left ureter.

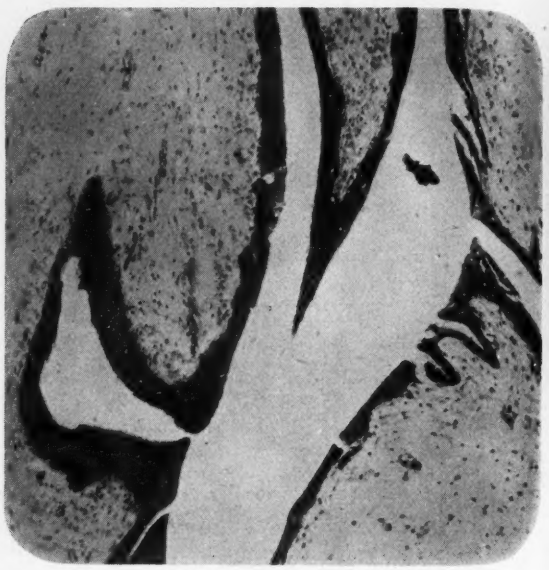

FIG. 309.—Same case. Microphotograph of papilloma, showing œdema of the fronds. (× 60.)

TREATMENT

The only rational treatment is complete uretero-nephrectomy. This can usually be performed in one stage under spinal anæsthesia quite safely. On rarer occasions, because of absence or disease of the opposite kidney, it may be essential to preserve that on the affected side. In such cases, depending on the site of the tumour, a segment of ureter can be removed and the end transplanted into the bladder or renal pelvis, or a permanent nephrostomy established. When the complete operation is performed there are two ways of dealing with the lower end of the ureter. It may be excised with a small portion of bladder wall or the extreme lower end may be left, its mucosa being destroyed by diathermy before ligature in order to destroy any neoplastic deposits in it. During this stage it is very important to avoid contamination of the pelvic cellular tissues with neoplastic cells.

X-ray therapy before and after operation is probably of little permanent value.

CASE REPORTS

Case 1.—**Simple papilloma of the ureter—Diagnosed by intravenous urography and cystoscopy—Treated by nephro-ureterectomy—Recovery.—**

Male, aged 25 years, complained of pain in the left renal area for fifteen months. Never very severe. Occasional painless hæmaturia for the last nine weeks. Examination: general condition good. No physical signs on complete clinical examination.

Urine.—Microscopical examination showed some red blood-cells.

Plain X-ray Examination showed no evidence of an opaque calculi.

Intravenous Urography.—Both kidneys were seen to have good function and normal caliceal outlines. There was some dilatation of the lower part of the left ureter, which had a curious mottled appearance (*Fig.* 308).

Cystoscopic Examination.—A papilloma could be seen protruding from the left ureteric orifice.

OPERATION.—Performed on June 19, 1940, under spinal anæsthesia. Complete uretero-nephrectomy, except for the lowest quarter of an inch of the ureter, was carried out. The mucosa of the lower end of the ureter was destroyed with diathermy before it was

ligated. The patient made an uninterrupted recovery, except for the leak of urine through the abdominal wound for a few days. To date there has been no recurrence of the neoplasm.

PATHOLOGY.—The specimen, consisting of the left kidney and ureter, was fixed before examination. In the lower third of the ureter there was a pedunculated papilloma (*Fig.* 310). It had an appearance

FIG. 310.—Same case. Coloured drawing of specimen. The lower part of the ureter is drawn separately slightly magnified. Although the diagram does not show it, the whole of the ureter, renal pelvis, and calices were carefully examined for evidence of papilloma, but none was found.

resembling an acorn. Although there was no intussusception of the ureter, it was obvious by the position of the attachment of the tumour that it could only prolapse through the ureteric orifice by such a process. The tumour had a more solid appearance to the naked eye than the ordinary papilloma. Careful examination, however, showed that this appearance was due to swelling and œdema of the fronds, probably caused by constriction of the base of the tumour by the ureter. Microscopical examination (*Fig.* 309) showed the structure of a simple papilloma with œdema of the fronds.

Case 2.—**Carcinoma of the ureter secondary to carcinoma of the kidney—Diagnosed by retrograde ureterogram—Treated by ureteronephrectomy.—**

Male, aged 57. Eight weeks' history of intermittent hæmaturia, pain in the left iliac fossa, frequency of micturition, and loss of weight.

ON EXAMINATION.—General condition fair. Abdomen, lower pole of right kidney palpable; left kidney also palpable, surface irregular.

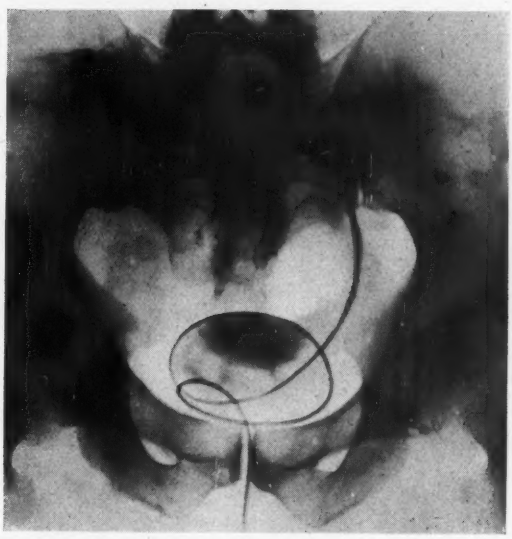

FIG. 311.—*Case* 2. Plain radiograph showing the ureteric catheter obstructed at the level of the sacro-iliac joint.

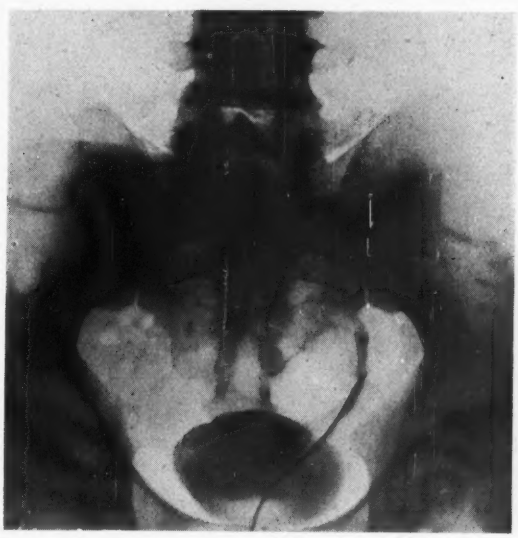

FIG. 312.—Same case. The catheter has been withdrawn a short distance, and the urogram confirms that there is obstruction at the level of the sacro-iliac joint.

Urine.—Contained obvious blood.

Plain X-ray Examination.—No opaque calculi, but very definite enlargement of left kidney outline.

Intravenous Urography.—Good function on the right side and normal caliceal outlines. No excretion on the left side in two hours.

Cystoscopy.—Bladder normal. Various sizes of ureteric catheters were passed up the left ureter, but all obstructed at 14 cm.

Retrograde Ureterogram showed an irregular obstruction of the ureter at this level (*Figs.* 311, 312).

OPERATION (June 19, 1939).—Spinal anæsthesia. The ureter was exposed extraperitoneally through an oblique left iliac fossa incision. A hard nodule was found in the ureter just below the level of the sacro-iliac joint. The ureter was ligated some distance below this and mobilized as high up as possible. There were numerous hard neoplastic deposits near it. The wound was closed and the operation completed through the ordinary oblique kidney incision. There was much extrarenal growth. As much of this as possible, together with the perinephric fat, was removed.

Patient had an uninterrupted convalescence, but in spite of deep X-ray therapy died four months after leaving hospital from recurrence.

PATHOLOGY.—The kidney, although it has retained its general shape, has been entirely replaced by hard white neoplasm (*Figs.* 313, 314). There is no evidence of involvement of the calices or pelvis on careful dissection. At the upper pole there is one large boss of extrarenal growth. In the lower part of the ureter there is an annular malignant deposit about ½ in. long, and about 1½ in. above this a hard deposit attached to the external wall of the ureter. Microscopical examination of the kidney and ureteric neoplasms shows an undifferentiated carcinoma

FIG. 313.—Same case. Drawing of the specimen, showing a marked boss of extrarenal neoplasm, a deposit in the lower ureter, and another one attached to the wall of the ureter about its middle.

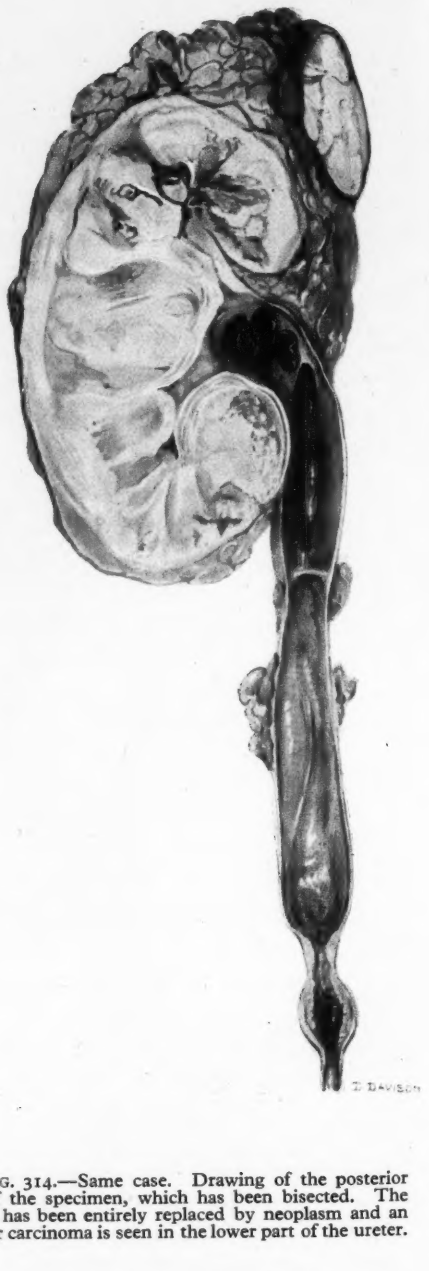

FIG. 314.—Same case. Drawing of the posterior part of the specimen, which has been bisected. The kidney has been entirely replaced by neoplasm and an annular carcinoma is seen in the lower part of the ureter.

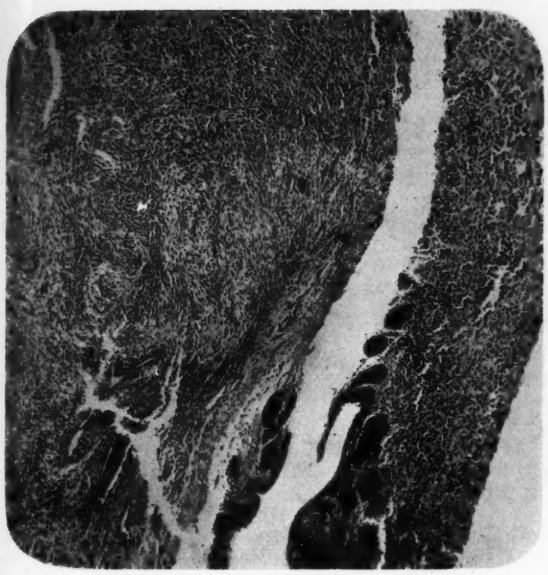

FIG. 315.—Same case. Microphotograph showing a neoplastic deposit of the wall of the ureter. (× 50.)

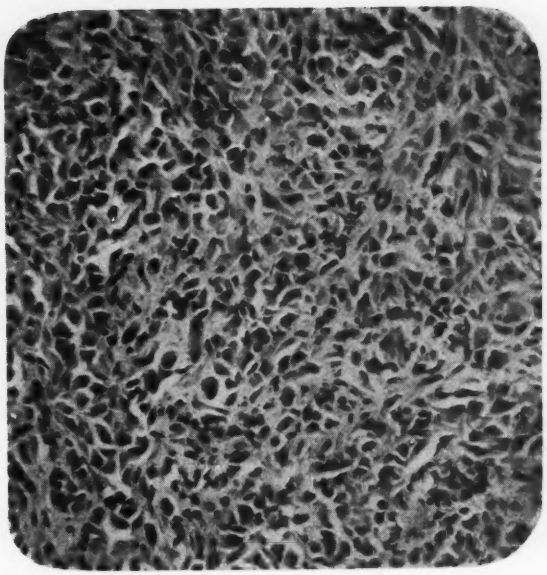

FIG. 316.—Same case. Microphotograph showing the large flattened type of cells of which the carcinoma consists. (× 200.)

growing mainly in diffuse scirrhous form. The cells are often spindle shaped and contain little lipoid. Although the appearances (*Figs.* 315, 316) suggest a transitional-cell epithelioma derived from the renal pelvis rather than a carcinoma of the tubules, the complete absence of any macroscopical evidence of involvement of the calices or pelvis rather discounts this. It is most probable that the tumour is a carcinoma derived from some renal tubules lined by flattened epithelium, as, for instance, the descending loop of Henle.

SUMMARY

1. Ureteric neoplasms both primary and secondary are rare.

2. They can be accurately diagnosed by patient investigation.

3. Radical surgical treatment is the only one with any chance of success.

4. Two cases are reported and the literature reviewed.

My thanks are due to Mr. H. H. Rayner, under whose care these cases were admitted to hospital, for permission to include them in this paper.

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THE HISTOLOGICAL CONDITION OF A NERVE AUTOGRAFT IN MAN

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WINGFIELD-MORRIS ORTHOPÆDIC HOSPITAL, AND THE DEPARTMENT OF ZOOLOGY AND COMPARATIVE ANATOMY, OXFORD

INTRODUCTION

ALTHOUGH useful recoveries have been reported after autogenous nerve-grafting in the hand (Bunnell and Boyes, 1939), the facial canal (Duel, 1933; Collier, 1940), and to some extent in the nerves of the limbs (Medical Research Council, 1920; Foerster, 1929), the pessimistic views held by most British observers at the end of the last war still prevail (Platt and Bristow, 1924). However, the need for some means of making good a large defect in a peripheral nerve remains, and the success that has attended experimental nerve-grafting (Huber, 1920; Bentley and Hill, 1936; Sanders and Young, 1942) suggests that improvements in technique may yet bring this method, which at any rate in theory has so much to commend it, into the field of practical surgery.

Grafts of the fascial tube type examined histologically by Stopford (*see* Platt, 1920) were apparently converted at least partly into fibrous tissue.* If this is the fate of all grafts then the failure of the method is absolute and nothing more can be done. On the other hand, any evidence showing that appreciable regeneration can occur through a human graft should be studied with care, for it may indicate the direction in which success will ultimately be found. This is our reason for reporting the present case, in which an autograft had to be removed because the stumps with which it had been joined were too badly damaged to allow recovery. This misfortune has provided an opportunity for showing that grafted pieces of nerve remain alive and provide a pathway for the growth of nerve-fibres.

CASE REPORT

May 29, 1940: R. M., a soldier of 26, was crossing the Channel from Dunkirk when a bomb struck the ship and a splinter passed through the soft tissues of his right arm. The median nerve was divided at a point 10 cm. above the medial epicondyle, but there was no bony or vascular injury.

May 30: Wound excised and drained at a hospital in Kent. There was moderate sepsis.

June 17: *Admitted* to the Wingfield-Morris Hospital.

July 14: Wounds healed. There was no retained foreign body and nothing of special note in the neurological condition apart from division of the internal cutaneous nerve of the forearm as well as of the median.

Although there had been some anxiety about the circulation in the arm when the patient first landed in England, we found no evidence of circulatory abnormality.

1ST OPERATION (September 14).—The median nerve was exposed under local anæsthesia and found to run into an enormous mass of scar tissue which involved skin, subcutaneous tissues, the nerve, brachialis, and triceps.

State of the Nerve: (*a*) *Central part* of the trunk—healthy; (*b*) *The lesion*—scar 4·4 cm. long, exceedingly hard, narrowed at one point to 8 mm. × 5 mm.; (*c*) *Peripheral trunk*—nerve yellow, elastic, and firmer than a normal degenerate nerve.

Electrical stimulation showed that a few sensory fibres had passed through the scar and grown down the peripheral trunk for a distance of at least 2·5 cm., but the patient's responses were not consistent, and it was decided to perform a resection and suture on the assumption that the nerve had been completely divided.

At this stage a change was made to general anæsthesia. The nerve was freed from the upper third of the arm down to the elbow, but mobilization was poor because of the fine scar tissue that enveloped the lower part of the nerve. But even so the mobilization was more timid than it should have been, and the nerve was not freed extensively in the antecubital fossa. After the resection of 3·5 cm. of scar tissue nerve-bundles were exposed proximally and distally. The central bundles were fairly good, but in the light of later experience one must confess that the peripheral cut surface showed far too much fibrous tissue.

It was then found impossible to get the nerve-ends together without great tension, and a nerve-graft was therefore performed, using three cables from the internal cutaneous nerve of the forearm, which were secured in good position with a fibrin coagulum (Young and Medawar, 1940).

It must at once be admitted that this was a poorly executed operation. Examination of the tissue removed showed that resection had been inadequate.

HISTOLOGICAL REPORT.—The piece of tissue excised at operation was fixed in formol, embedded in paraffin, and cut into serial longitudinal sections. These were stained by the Bodian method for axons (Bodian, 1936), with Mallory's or Masson's trichromes, or a combination of these with the Bodian method.

The piece was 2·8 cm. long after fixation. The upper 10 mm. consisted of recognizable but damaged

* Though in 1921 Stopford reported a case operated on by Platt, of a graft from the radial nerve, presumably a single strand, used to bridge a gap in the ulnar, in which there were undoubted signs of conductivity thirty-two months after operation.

nerve (the '*central stump*'). The next 15 mm. was dense *scar tissue*, and in the distal 3 mm. there were again some traces of nerve (the '*peripheral stump*') (*Fig.* 317).

Central Stump.—The central stump occupies the greater part of the thickness of the piece in the region where it occurs. Its extent at the most proximal point has been mapped by projection (*Fig.* 318). In this reconstruction only the clearest bundles have been included, and there are many other regions occupied by scattered irregular bundles. Even at the proximal cut surface the nerve is highly modified, as if it had undergone degeneration and re-innervation. All of the fibres are very small and probably little myelinated. There has been much proliferation of Schwann nuclei and infiltration of macrophages (*Fig.* 319, A), and there is much interfascicular fibrosis. Many of the bundles do not run straight, and are criss-crossed, even at the proximal surface.

This surface would certainly provide new fibres for growth into the graft, but it is not a normal stretch of nerve, and the prognosis would not be good on this account alone, even if all other factors were favourable.

Scar.—The scar region shows that there has been complete interruption of the nerve, a piece of skin having been driven right across it. There are several areas occupied by large masses of live hair follicles, especially across the distal end of the central stump (*Fig.* 317). Many other hair follicles lie away from the main masses, and are growing, with live sebaceous glands, actually within the nerve. Most of this impacted hair has been removed by the operation, but probably not all, for there are groups close to the line of excision at the central end (*Fig.* 320), and some hairs actually at the cut surface of the peripheral end (*Fig.* 321).

Peripheral Stump.—Only a very few definite bundles can be seen: they have been reconstructed in *Fig.* 322. They do not occupy nearly enough area to correspond to the central stump.

Conclusions.—Considering (1) the absence of good peripheral bundles, and (2) the poor state of the central stump, it was nearly certain that no recovery could take place. Resection at the lower end of the graft and fresh union might give some partial recovery, but the prognosis would still be poor unless the second factor was also dealt with.

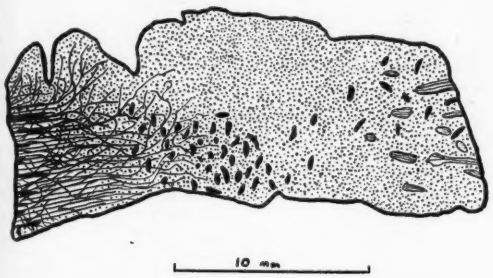


FIG. 317.—Sketch of longitudinal section of the whole piece of tissue removed at operation, Sept. 14, 1940. The central stump is to the left. The black rods indicate hairs.

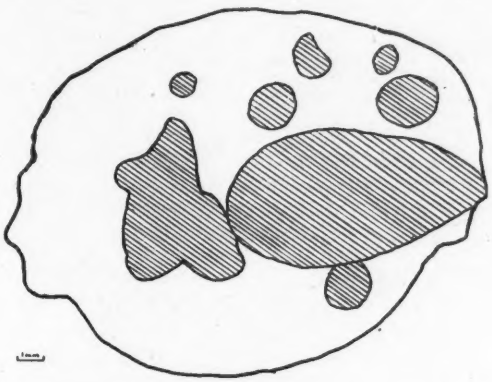
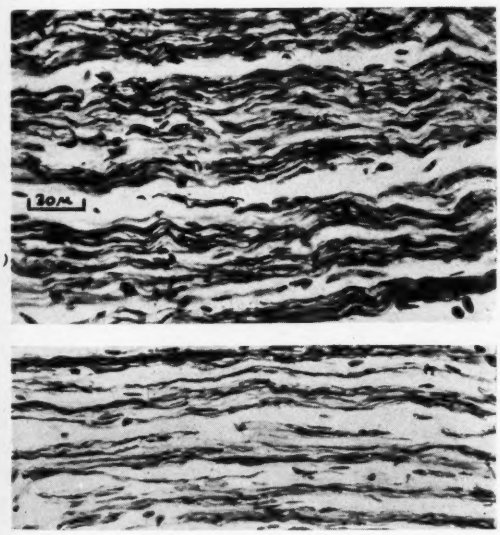
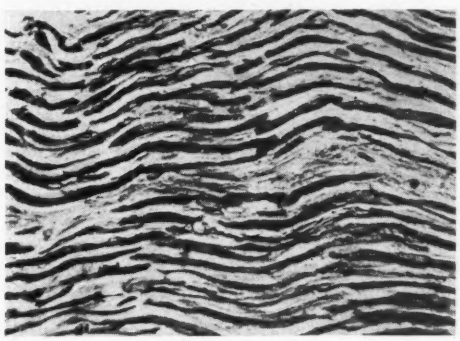


FIG. 318.—Reconstruction of the central end of the piece removed Sept. 14, 1940. The hatched areas represent bundles of fibres. Some of the rest of the area is occupied by small scattered bundles.



A



B

FIG. 319.—A, Longitudinal section of abnormal nerve-bundles at central end of piece resected on Sept. 14, 1940. Bodian's method. B, Section of a normal nerve for comparison.

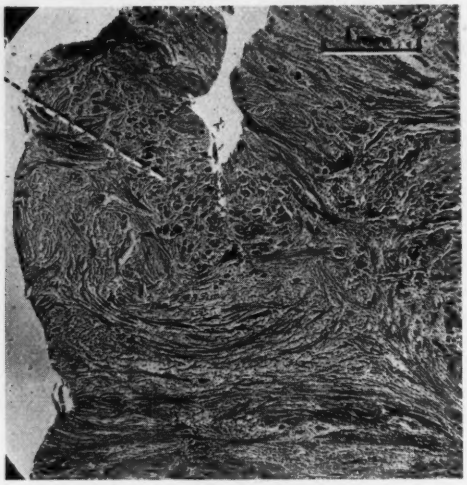


FIG. 320.—Longitudinal section of central end of piece removed. Low-power view. Note very irregular bundles of nerve-fibres and masses of hair follicles (H). Bodian's method.

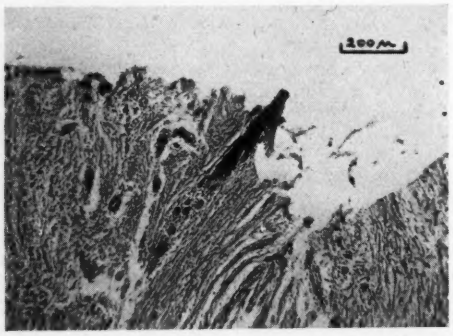


FIG. 321.—Live hair protruding at the distal surface of the piece removed. Hæmatoxylin and eosin.

SUBSEQUENT PROGRESS.—In April, 1941, there was no sign of recovery in the common flexor group of the forearm; in view of this it was decided to explore the nerve again and attempt an end-to-end suture.

2ND OPERATION (April 16, 1941).—Under general anæsthesia. Incision from the coracoid process to the head of the radius. The scar tissue round the nerve was less dense than at the last operation, but sufficient to make dissection difficult. The pectoralis major was divided up to the coracoid process and the nerve traced downwards from the lower border of pectoralis minor. Below, the nerve was followed upwards from the margin of the pronator teres, the deep head of the muscle being divided and all branches of the nerve stripped up for about 5 cm.

State of the Nerve.—

a. Central: 12 cm. above the epicondyle the nerve became moderately fibrous, and this continued peripherally for 4 cm. The trunk was firmer than normal, and 11 mm. in diameter. The diameters of the healthy nerve above were 9 mm. × 6 mm.

b. The graft region: This was 7·5 cm. long, and rather like an hour-glass in shape (*Fig.* 323), the diameter of the proximal swelling being 16 mm., the waist 11 mm., and the distal swelling 14 mm. At no point was the grafted segment densely firm, and the waist was little firmer than normal nerve.

c. Peripheral: Colour yellowish, with small varicose veins on its surface; slightly firmer than normal, diameter 7 mm. × 5 mm.

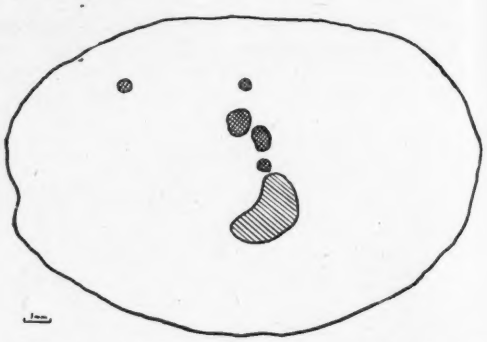


FIG. 322.—Reconstruction of the peripheral end of the piece removed Sept. 14, 1940. The cross-hatched areas are good peripheral bundles. The single-hatched area is a doubtful bundle.

Results of stimulation: Bipolar faradic stimulation —no response.

Results of mobilization:

Freeing nerve proximally	gain ..	2 cm.
„ „ distally	„ ..	2 cm.
Flexing elbow 90° ..	additional	4 cm.
„ „ from 90° to 140°	„	2 cm.
Adduction and internal rotation	„	3 cm.
	Total gain ..	13 cm.

Procedure.—The grafted segment of nerve was resected, the piece removed being 9 cm. long. Healthy bundles were exposed above and below. As there was an enormous bed of scar tissue it was found best to fold this scar tissue over on itself and transfer the nerve to a most convenient groove that was found between the biceps and brachialis—and this was actually the shortest route for the nerve to take. The wound was then closed in layers except at the site of nerve suture. Good closure was obtained by the insertion of ten No. 1 silk sutures. Wound closed: arm put in plaster with elbow flexed 130°.

HISTOLOGICAL EXAMINATION OF GRAFTED TISSUE REMOVED.—The whole piece was fixed in formol-saline. The most proximal and distal regions were

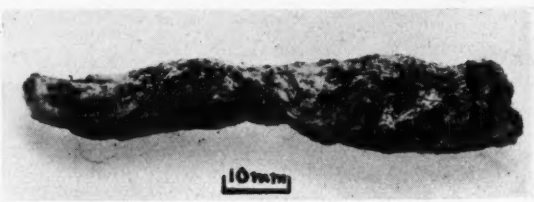


FIG. 323.—Photograph of the graft when removed on April 16, 1941. Central end to the left.

cut off after fixation, and mordanted with potassium bichromate, embedded in paraffin, cut in transverse section, and stained by Weigert's method.

Central End of Resected Tissue (representing suture point of April 16, 1941).—The sections from the central end show good nerve-bundles (*Fig.* 324, A). There is no complete epineurium around them, and the perineurium shows considerable thickening. But in only a few places does this scarring extend into the

endoneurium, which is therefore mostly normal. However, many, if not all, of the bundles have undergone degeneration and subsequent re-innervation, as is shown by the multiplicity of Schwann nuclei in the nerve (*Fig.* 325). Many medullated nerve-fibres of all sizes are present, and give reason to suppose that the stump is in a condition to allow of recovery.

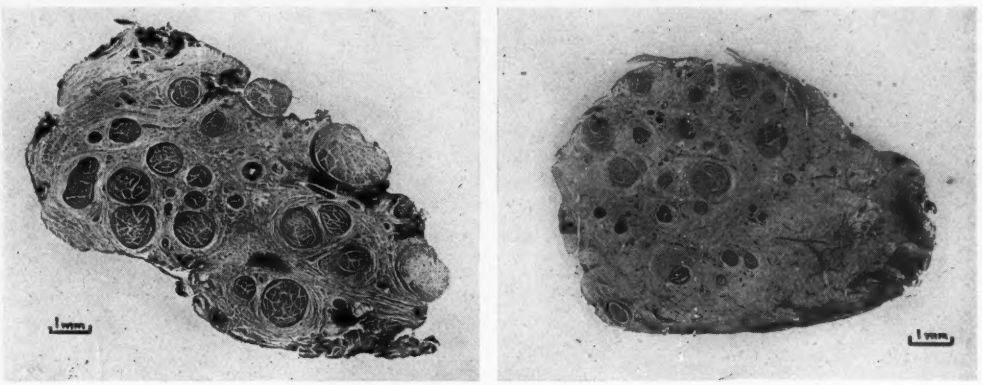


FIG. 324.—Cross-sections at different levels of the the piece removed at operation of April 16, 1941. A, Near central suture point (Weigert); B, At middle of the graft (hæmatoxylin and eosin); C, Near distal suture point (Weigert).

Peripheral End.—This also contains many good bundles (*Fig.* 324, C), though with some interfascicular fibrosis. The endoneurium is mostly free from scarring, and contains many normal Schwann bands

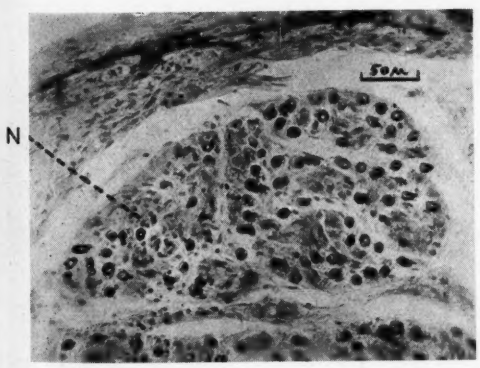


FIG. 325.—Transverse section of a nerve-bundle near the central suture point, showing medullated nerve-fibres and many tubes containing proliferated Schwann nuclei (N). (Weigert, alum-carmine.)

of degeneration (bands of von Büngner) (*Fig.* 326). In some of the bundles there are also considerable numbers of small medullated nerve-fibres, which must have crossed the graft and reached the peripheral stump in spite of all the difficulties. Since, as has been described, only a few of the bundles of the peripheral stump reached up to the surface that was sutured to the graft, it is not surprising that these medullated fibres are found in only some of the peripheral bundles.

Histology of the Graft.—

After removal of the most proximal and distal pieces, the remainder of the tissue excised, including the graft and the central and peripheral junctions, was divided in two for convenience, embedded in paraffin, and cut in both transverse and longitudinal

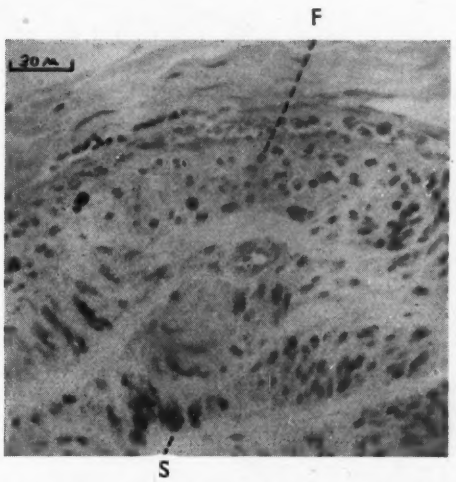


FIG. 326.—Transverse section of a nerve-bundle near the peripheral suture point, showing many Schwann nuclei (S) and small medullated fibres (F).

section. The sections were stained variously by the silver methods of Bodian (1936) and Holmes (1942), by trichrome stains, and by hæmatoxylin and eosin.

The upper graft junction: The longitudinal sections stained by the silver methods show that although the

central bundles used for suture on Sept. 14, 1940, were far from normal, yet they have made quite a good junction with the upper end of the graft. *Fig.* 011 shows that there is some criss-crossing of fibres at the point of union, but this occurs at most if not all nerve junctions, and considering the conditions the junction may be regarded as a successful one. Certainly it shows that good connexions between graft and central stump can be obtained.

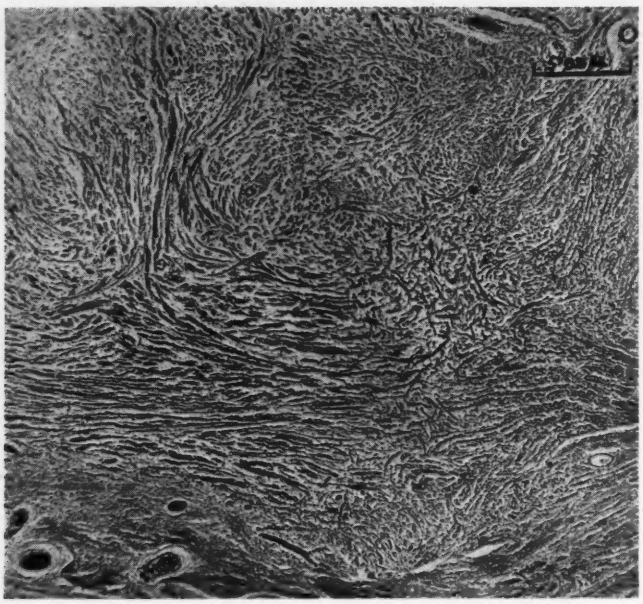
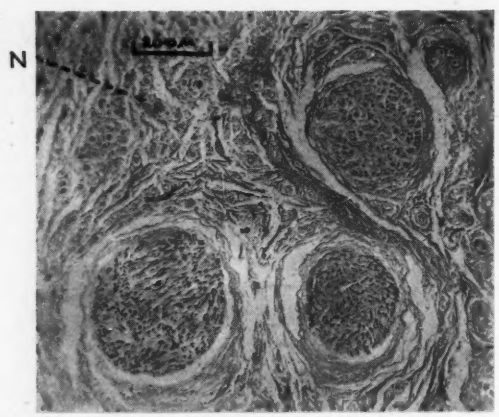
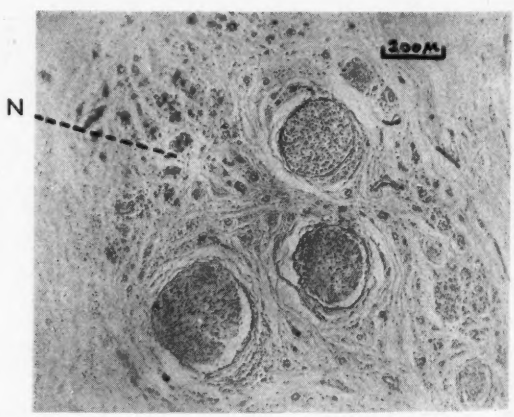


FIG. 327.—Longitudinal section of the point of union of the central stump (on left) with a grafted bundle. The fibres in the central stump run very irregularly, but quite a good union has been made. Bodian's method.

State of the graft itself: Transverse sections taken at the middle of the graft show that the nerve-bundles have been very well preserved (*Figs.* 324, B, 328, 329). by fibrous tissue, but this does not appear to be dense or likely to contract down on to the bundles. Indeed it is providing a pathway for the outgrowth of new bundles of nerve-fibres, for between the main bundles in the graft there are to be seen very many small ones which certainly represent new growth (*Figs.* 328, 329). Moreover, the connective tissue round the bundles has become to some extent orientated to make a perineurium rather than a dense avascular scar (*Fig.* 328).

The grafted bundles themselves have the appearance of normal peripheral stumps as seen after nerve





FIGS. 328, 329.—Transverse section of grafted bundles at the middle of their length. *Fig.* 328 is stained with silver (Holmes) and shows nerve-fibres. *Fig.* 329 stained with hæmatoxylin and eosin shows the nuclei. Note the many new bundles (N) in addition to those grafted.

They do not appear to be collected into groups corresponding to the original number of strands of nerve used. There is, of course, no common epineurium. The space between the bundles is occupied section. They contain abundant elongated Schwann nuclei (*Fig.* 330), showing that proliferation of these cells proceeds normally in the graft after the operation. The remains of the original myelin in the nerve have

been completely removed. There are no signs whatever of any abnormal cellular reactions or necrosis in any part of the bundles. The blood-supply consists of vessels of normal appearance, but whether they contain any part of the original vessels cannot be determined.

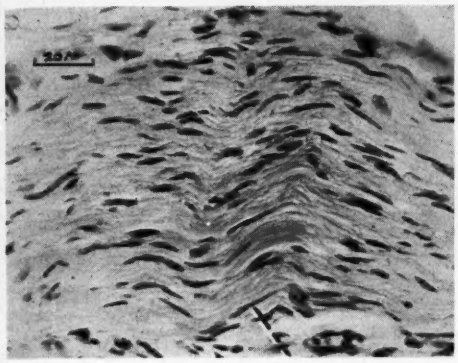
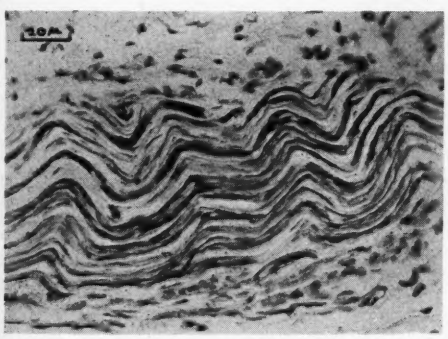
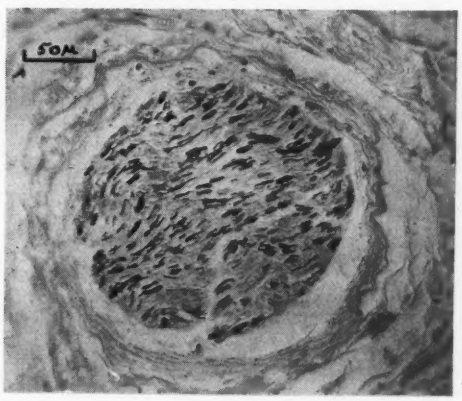


FIG. 330.—Longitudinal section of grafted bundle to show Schwann nuclei (S). (Hæmatoxylin and eosin.)

The grafted pieces are full of new nerve-fibres (*Figs.* 331–333). These are of various diameters, none of them quite as large as in a normal nerve, but this is not to be expected after a time as short as four months. The stains used do not allow of a proper demonstration of the myelin present, but the outline of such myelin as remains after paraffin embedding can be shown by a variant of the method of Holmes, and it is clear that some of the fibres are well medullated, and show Schmidt-Lantermann incisures.





FIGS. 331, 332.—Longitudinal and transverse sections of grafted bundles showing axons of medullated fibres. (Holmes' method.)

The lower graft junction: Since only a few bundles of the peripheral stump reached to the graft, the majority remain separated from the distal end of the graft bundles by a gap of several millimetres. The graft bundles which are not in contact with the peripheral stump spread out at their lower ends (*Fig.* 334). Fibres sprout from them in all directions through the scar, some reaching the peripheral stump and forming the groups of medullated fibres which were found there. There is nothing to suggest that the lower junction would not have been satisfactory if normal peripheral bundles had been present.

DISCUSSION

This case leaves no doubt that under suitable conditions the tissues of transplanted nerves can remain alive and become directly incorporated in their new position. The bundles seen are essentially similar to those found in a normal peripheral stump, and are too large to have been laid down by replacement of the grafted tissue. There is no indication that the graft would have been destroyed at some later time, and indeed as explained on p. 382 the connective tissue around the bundles appears to be organized as a sort of perineurium. Yet the position was not very favourable for the graft, in that the bed was heavily scarred, and in particular the poor state of the stumps must have prejudiced the chance of a quick vascularization of the graft from the ends. It is possible, though not proved, that the success of such grafts depends on the fact that they are made up of thin strands. Bielschowsky and Unger (1917) and Maccabruni (1911) report that in dogs necrosis took place at the centre of thick grafts, but the subject has never been thoroughly investigated.

A graft thinner than its host nerve can hardly give more than a partial recovery. In this case where three strands of cutaneous nerve were used, the cross-sectional area of nervous tissue in the graft was only 30 per cent of that in the central stump, but 70 per cent of that in the peripheral stump. Even if the graft had been united with satisfactory nerve-bundles, it is doubtful whether a really good recovery could have been made, but some improvement might have been expected. Where cable grafts are used every effort should be made to provide an area of nerve approaching to that of the stumps with which the graft is united.

That autografts can survive and become innervated agrees with the results of many animal experiments (Huber, 1920; Bentley and Hill, 1936; Sanders and Young, 1942), and with

the recoveries reported, as mentioned in the introduction, after the use of autografts in man. There have certainly also been many failures, possibly mainly due to the fact that the grafts were too thin to carry an adequate number of fibres to the periphery (as in the case reported

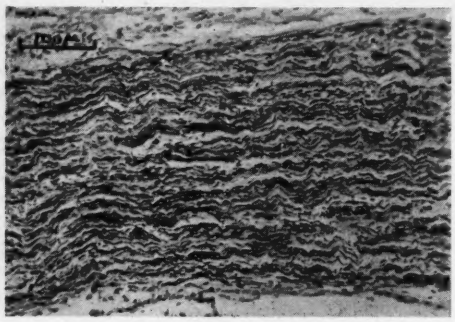


FIG. 333.—Longitudinal section of grafted bundle. (Bodian's method.)

by Stopford, 1921). And the present case shows how important it is to be sure, before reckoning a graft as a failure, that it has been joined to stumps that are in a state themselves to allow of recovery.

SUMMARY

1. A cable autograft placed to bridge a lesion in a human median nerve was removed seven months after operation and examined histologically.

2. The transplanted tissues had survived, and Schwann cell proliferation and the removal of myelin had proceeded normally.

3. New nerve-fibres had entered the graft and acquired myelin sheaths. The appearance of the transplanted bundles was therefore essentially similar to that of a normal peripheral stump after nerve section.

This investigation was assisted by a grant from the Rockefeller Foundation.

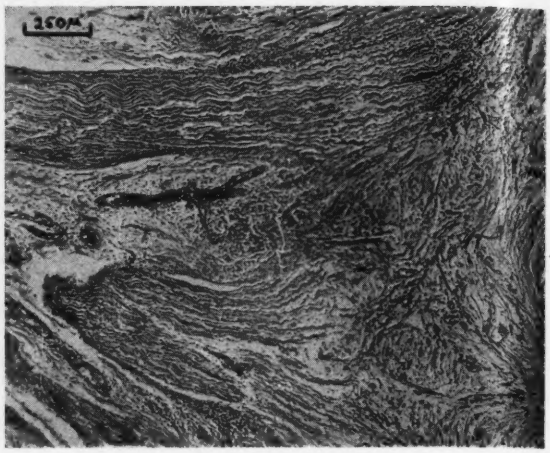


FIG. 334.—Longitudinal section of lower end of the graft, to show fibres emerging from the grafted bundles. (Bodian's method.)

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TRAUMATIC PERFORATION OF THE INTESTINE AS A COMPLICATION OF INGUINAL HERNIA

By JOHN BRUCE
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THE presence of an inguinal hernia favours rupture of the intestine when trauma is applied to the intact abdominal wall. This association has been frequently commented on in the Continental medical press, and several explanations have been proposed to account for it. The case reported here affords striking support to one of the theories advanced.

CASE REPORT

HISTORY.—A farm labourer, 63 years of age, was admitted to hospital in the early hours of the morning with acute abdominal pain. At 8 p.m. on the previous evening, he had stumbled after unyoking his horse, and fallen heavily on the shaft of a farm-cart, the force of the impact being received on the lower part of the abdominal wall. Almost at once, he complained of severe pain in the right groin, and this rapidly spread over the whole lower abdomen. It was still present and considerable, at the time of admission. A severe degree of shock had been noticed immediately after the accident, and on his admission after a journey of fifty miles, he was gravely ill.

He had suffered from "stomach trouble" for many years, his principal complaint being lower abdominal pain and constipation. Bilateral inguinal herniæ had also been present for some years, though they gave rise to no symptoms, and indeed, only appeared on exertion.

ON EXAMINATION.—He was pale and very collapsed. The temperature was subnormal, and his pulse-rate 120 per minute. The abdomen was slightly distended, and the lower half of the abdominal wall immobile. Tenderness was elicited over the right lower quadrant and the right inguinal region. The muscles of the abdominal wall were rigid over the same extent.

A diagnosis of rupture of the intestine was made, and after a period of resuscitation, he was submitted to operation.

AT OPERATION.—The abdomen was opened by a right lower paramedian incision. A small quantity of turbid fluid escaped when the peritoneum was entered, but there was no very obvious degree of peritonitis. A mass of intestine immediately presented at the wound, with the coils matted together to such an extent that their separation was quite impossible. The mass was about a foot long, and involved a considerable part of the ilium; it was obviously a very old lesion, probably tuberculous in nature.

A small perforation was present on one of the loops of the mass. The edges were irregular and the aperture gaped open (*Fig.* 335). The ideal treatment would have been to resect the whole affected segment of intestine, but the man had taken the anæsthetic badly, and his condition was giving rise to anxiety. Accordingly, the perforation was rapidly closed by a Lembert suture, and the abdomen closed with through-and-through sutures around a drainage tube. Despite the usual post-operative measures, the man's condition rapidly deteriorated, and he died six hours after operation.

FIG. 335.—The matted loops of intestine, showing the small gaping perforation.

POST-MORTEM FINDINGS.—The autopsy findings were of considerable interest. The peritoneal cavity contained a few cubic centimetres of blood-stained exudate, mainly in the pelvis. The visceral peritoneum was congested, and there was some congestion, though not marked, of the parietal peritoneum.

Hernial sacs were present in both inguinal canals. On the left side the sac was small, only admitting the tip of a thumb; its serous surface was not noticeably congested. On the right side the sac had a somewhat larger abdominal ostium, with a firm, almost rigid neck. The sac itself was thick-walled, and its serous surface presented a picture of intense congestion. The suddenness of the colour change at the ostium was very striking (*Fig.* 336).

DISCUSSION

Rupture of the intestine in cases of hernia may, under certain circumstances, be readily explained. Ill-advised taxis, for example, may lead to rupture of an incarcerated hernia, and the reception of a direct blow either directly on a hernial protusion, or indirectly on an ill-fitting truss, may have a similar effect. In the present case the rupture followed the application of force to the abdominal wall at a point remote from the inguinal canal, and this type of injury is at first sight not readily understood.

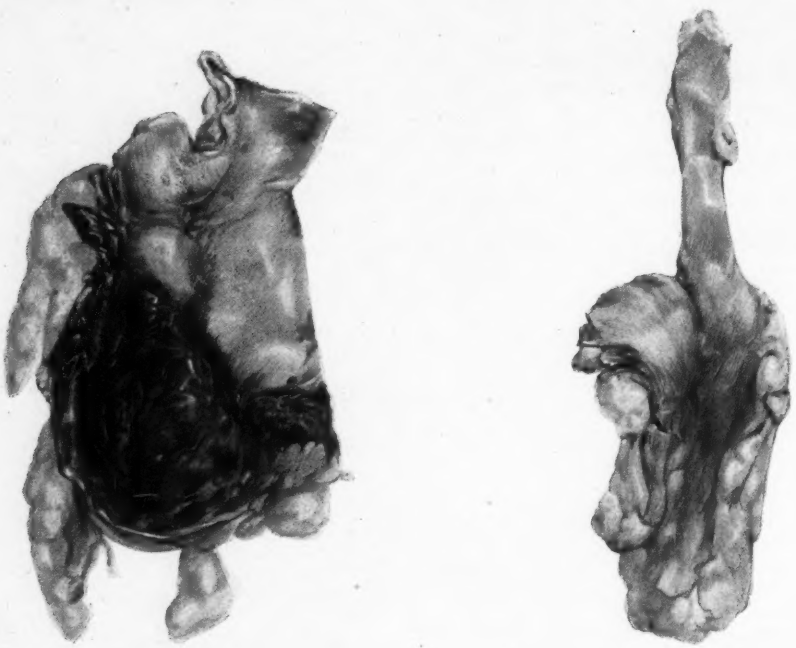

FIG. 336.—The hernial sacs after removal post mortem. The contrast between the congested right sac and the normal left sac is well shown, as is the abrupt transition between the general peritoneum and the inflamed peritoneum at the neck of the sac.

This particular form of intestinal rupture is not excessively rare. Thirty cases had been reported up to 1937, when Aird added three cases and reviewed the Continental literature on the accident. So far as I can find, no further cases have been described, though it is more than probable that the presence of the hernia or even its significance, may have escaped notice in cases of traumatic subcutaneous rupture of the intestine. The writer has encountered a case of this sort recently, in a soldier brought up before a Medical Board. In this man, rupture had followed a kick on the lower abdomen, and successful laparotomy had been carried out some months before. A right inguinal hernia was present at the time of the examination, but it had not been observed before the operation. While it is likely that the hernia was in existence at the time of the accident, and contributed to the sequence of events, it is impossible to be quite certain, since rupture of the intestine may occur without hernia. This has been the case in a patient of my own who ruptured his jejunum when the wheel of a vehicle passed over his abdomen; and I have seen—again at a Medical Board—a soldier who had been operated upon for ruptured intestine, and in whom a hernia was absolutely excluded by my own examination.

In addition to blows on the abdominal wall rupture of the intestine has followed violent hyperextension of the trunk, and has been claimed to follow sudden contraction of the abdominal wall, as in standing up quickly or rising from bed.

The lesion is most commonly situated in the lower ilium, and lies close to the abdominal inguinal ring—so often is this the case, indeed, that an inguinal herniotomy approach is advised, instead of laparotomy, by certain German writers. In practically all cases the hernia itself has been previously reducible; in many it has actually not been 'down' at the time of the accident.

The most ingenious explanation of the rupture is that advanced by Bunge, who attributed it to a sudden rise in intra-abdominal pressure. Increased pressure within the abdominal cavity must follow violent hyperextension of the trunk, and falls on the flat belly, blows on the abdomen, and sudden and severe contraction of the muscles of the anterior abdominal wall. It is, therefore, a common factor in all the varieties of accident which have precipitated intestinal perforation. The increase in the intra-abdominal tension is accompanied by a rise in the pressure within the

bowel, but when the abdomen is normally closed off, this is of no significance because it is equalized by the pressure outside the bowel.

When the abdominal cavity is not completely shut off, but has an outlet into the inguinal canal, rupture is possible in the loop of intestine overlying the inguinal ring, or contained in the hernia, since the hernial sac does not share in the general rise in intra-abdominal pressure. The increased tension within the loop is not equalized, and the unsupported part of the bowel bulges into the sac and bursts.

Fixation of an intestinal loop is believed by Aird to be responsible in the majority of cases. The bowel is fixed at the ileocæcal valve, on the one hand, and by the contraction of the muscles round the neck of the hernial sac on the other, so that it is tightly stretched. In these circumstances, it may be broken across by a force applied to the abdomen, or crushed between the external force and the posterior abdominal wall.

In the case reported here the post-mortem evidence affords striking support for Bunge's theory, in so far as there was intense congestion of the serous covering of the right hernial sac, which indicated a severe degree of irritation there. The rupture had unquestionably occurred at the mouth of the sac, so that the initial expulsion of contents has been into the sac. The peritoneum of the opposite sac, and the parietal peritoneum of the abdomen were at the time of death only mildly involved, and the contrast between the involved and uninvolved peritoneum was marked and striking. It is unlikely, as Aird says, that this explanation would cover all cases. That it does in fact occur, is demonstrated by this case.

SUMMARY

Rupture of a loop of intestine may be caused by indirect violence applied to the abdominal wall in cases of inguinal hernia. One of the causes of this complication is bursting of the bowel opposite the neck of the hernial sac; the rise of intra-intestinal tension finds the affected loop unsupported here because of the lower pressure within the hernial sac.

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GLOMUS TUMOUR

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A CASE of glomus tumour of an uncommon site (only one other such case appears to have been recorded) has recently come under my observation.

CASE REPORT

The patient, a man aged 38, stated that ten or twelve years previously he had a boil on the back of his neck, which was incised. Ever since, he had noticed a small swelling at the site of the incision, which very slowly increased in size and was always acutely sensitive to touch, mere contact with the collar provoking severe pain shooting up the back of the head.

On examination it was noted that the head was held stiffly in a slightly stooping position to avoid the friction of the collar, and that when the patient wished to look sideways he turned the eyes or the whole body. On the left side of the back of the neck, close to the middle line, was a slightly raised, blue, cystic globule looking like a small whortleberry embedded in the skin. On palpation, which was very painful, this small swelling was found to be the superficial part of a considerably larger firm, rounded tumour lying in the subcutaneous tissue.

A diagnosis of glomus tumour was made and the nodule was excised. Under novocain anæsthesia—which was not entirely successful in abolishing pain—an elliptical incision was made enclosing the cutaneous part of the tumour, and the mass was shelled out from the subcutaneous fat, and, with the attached ellipse of skin, removed.

The tumour measured 1·5 cm. × 1·0 cm. × 1·0 cm. The cut surface was of a pale greyish colour and resembled that of a lymphatic gland. The microscopical structure was that of a glomus tumour of predominantly angiomatous type (*Fig.* 337). The fairly thick fibrous-tissue capsule enclosed a tangle of vascular spaces lined by a single layer of flattened cells, and imbedded in a stroma composed of glomus cells and bundles of unstriped muscle-fibres running in all directions. Nerve-fibres could not be demonstrated.

It cannot be stated with certainty that any instance of glomus tumour was recorded before 1920, when Barré[6] published his case. The heterogeneous group of tumours to which the older writers gave the name of "painful subcutaneous tubercle" may have included examples of it, but as microscopical proof was lacking at that time it is impossible to say. It is noteworthy at least that in neither of its most characteristic clinical forms, the subungual and the subepidermal does the glomus tumour figure in the early literature. The elucidation of the nature of the tumour is due to Masson,[60,61,62] who first recognized its relation to the glomera of the skin.

Since Masson's papers were published a considerable number of cases has been put on record, chiefly in France and America. It has been possible to collect from the literature reports of 197 cases. Of these 24 have been rejected; in 9 the tumour was either not removed or not examined microscopically, in 3 the diagnosis was

for other reasons regarded as not proven, and in 12 the tumours were multiple (*see below*). On the remaining 173 and on the foregoing new case the following survey is based.

THE CUTANEOUS GLOMUS

Masson states that glomera were first recognized in 1837 by Berres, but it was not until 1852 that their structure became accurately known from the work of Suquet and of Hoyer. Later, detailed studies have been carried out, in particular, by Masson, Grant,[37] and Popoff.[75]

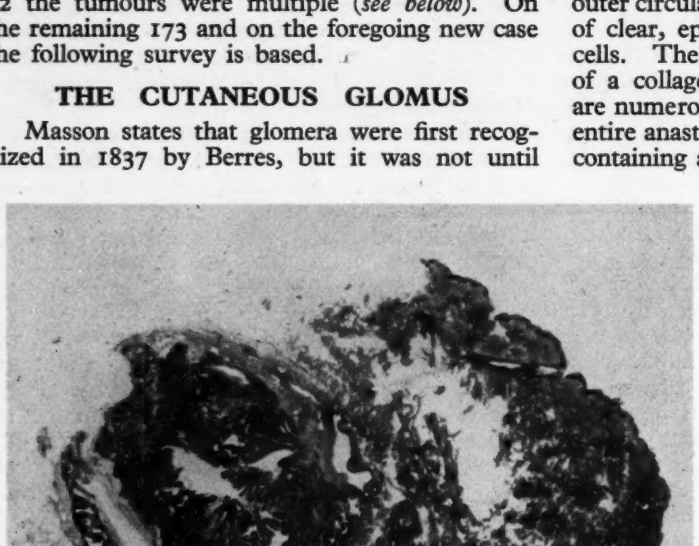

FIG. 337.—Section of tumour (low power).

Glomera have been found only in warm-blooded animals, in the 'exposed' parts of the skin (extremities of the limbs, ears, snout, tail), and in the erectile organs. In man they have been observed only in the hands and feet. They are situated in the deepest layer (stratum reticulare) of the corium; in rare instances they have been found within bone (the terminal phalanx) traversed by an artery bound for the skin.

The distribution in man is as follows: in the hand they occur in the nail-beds; the tips of the fingers and the palmar surface of the first, second, and third phalanges; the thenar and hypothenar eminences. In the foot the distribution is similar, and in addition they are found in the sole, near the heel. The numbers are very variously estimated by different authors; for instance, for the finger nail-bed Grant gives a figure of 501 per sq. cm., Popoff of 24, Masson of 10.

The glomus is a specialized anastomotic mechanism connecting a terminal arteriole with a primary venule.

The anastomotic vessel is S-shaped. The wall has an endothelial lining and two ill-defined muscular layers, an inner longitudinal and an outer circular. Among the muscle-cells lie groups of clear, epithelioid cells—the so-called glomus-cells. The outer zone of the anastomosis consists of a collagen network, in the meshes of which are numerous non-medullated nerve-fibres. The entire anastomosis is surrounded by fibrous tissue containing a plexus of collecting veins.

The function of the glomus is still uncertain. It is clear, at least, that the direct connexion between artery and vein offers a means of short-circuiting the capillary system, which may be of importance in the machinery of temperature-regulation, either local or general. Glomera do not develop until the end of the first month of life, and atrophy after about the age of 60.

The origin of the characteristic glomus-cells is likewise unknown. It has been suggested that they are an embryonic form either of smooth-muscle cells (myoblasts) or of vascular endothelium (angioblasts).

THE GLOMUS TUMOUR

Glomus tumours are composed of the tissues which make up the glomus, i.e., endothelium-lined vascular channels lacking an internal elastic lamina, smooth-muscle cells, nerve-fibres which (in contrast to Popoff's findings in the normal glomus) may be medullated as well as non-medullated, collagenous material, and, most important from the diagnostic point of view, glomus cells. These ingredients are jumbled together in a disorderly fashion and in varying proportions. Masson distinguishes four types of tumour according to the histological structure: the first is predominantly angiomatous; the second contains fewer vessels and a larger proportion of musculo-endothelioid stroma (the paucivascular type); the third consists largely of nerve-fibres (the neuromatous type); and the fourth comprises degenerative forms showing either simple œdema or hyaline and mucoid changes. Mulazzi[67] in a later paper gives a more elaborate classification.

From its point of origin in the deepest part of the skin the tumour may grow outwards towards the skin surface, inwards towards the subcutaneous fat, or in both directions. On its deeper aspect it shows a well-defined capsule. It may produce pressure-atrophy of bone when, as in the case of digital tumours, there is close contact, but it does not invade other structures or display any other malignant tendencies. Malignant tumours said to resemble glomus tumour in structure have been described. But in none of these cases is the evidence convincing, and in general it may be said that the glomus tumour is

essentially a benign growth. Local recurrence after operation has been reported several times,[52,53,63,67,89] but there seems no reason to doubt that it was due in each case to incomplete removal. Persistence of symptoms after removal of a subungual growth in a patient who was ultimately cured by excision of the entire nail-bed was presumably due to the same cause.[90]

The tumour is usually solitary; 13 cases of multiple tumours have, however, been reported. Because of the possibility of confusion with other conditions, the angiomatous leiomyomata in particular, all multiple growths have been omitted from this survey with the exception of the case reported by Picard,[72] in which the three small masses described seem to have been lobules of a single tumour rather than separate growths.

Incidence.—Glomus tumours in general appear to be slightly more common in males than in females, digital (including subungual) tumours decidedly more common in females than in males. They have been seen at all ages from 6 to 82. The sex and age distribution of the cases in which these details are given is shown in *Fig.* 338.

Location.—Though glomus tumours have been observed most often in places where glomera are normally found, they are by no means confined to these areas. It may be that glomera are distributed more widely than has been supposed, but so sparsely as to escape detection, or that they sometimes occur as heterotopic organs. The great majority of glomus tumours occurs in the limbs, especially the upper limb; elsewhere they are extremely rare. The anatomical distribution of the tumours is shown in *Table I.*

The exact location of the tumour in relation to the skin is mentioned surprisingly seldom in the published cases. Growing as it does from the corium, the glomus tumour should be anchored to the skin and inseparable from it on clinical examination. But expansion may take place chiefly downwards, so that the bulk of the tumour comes to lie in the subcutaneous tissue, and it may then be possible to demonstrate no connexion, or only the most tenuous bond, between it and the skin. Moreover, it would be absurd to assert dogmatically that glomera or glomus tumours cannot occur except in the skin. At all events, purely subcutaneous tumours have been reported, one by Masson himself.

Signs and Symptoms.—A glomus tumour is often visible. Except when it lies beneath the nail the tumour is also, as a rule, to be felt; occasionally it is too small or too deep to be palpable. It forms a firm, well-defined nodule, handling of which often causes the patient pain.

The outstanding symptom of a glomus tumour is pain, which may be aching or sharp in character—more often the latter—and sometimes has a burning quality. It is hardly ever absent; of the 173 patients only 3 made no complaint of pain. It is frequently of great severity, making the patient's life a burden. The pain usually occurs in attacks, which may start apparently spontaneously or may be brought on by some insult to the tumour, such as a blow or even a light touch, e.g., contact with the clothing.

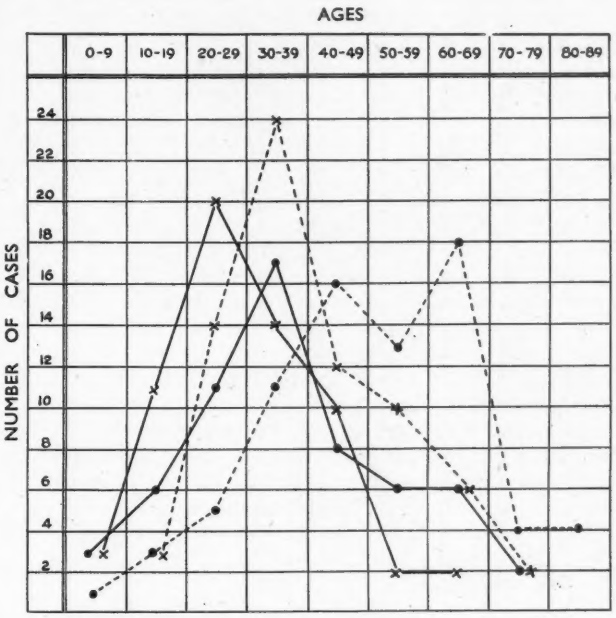

Fig. 338.—Sex and age distribution of glomus tumours. Male •; Female ×. The continuous line represents the age of onset; the broken line the age at which patient came under observation.

Table I.—Anatomical Distribution of Glomus Tumours

Site		Male	Female	Sex not stated
Upper limb	Shoulder and arm	13	3	3
	Elbow and forearm	16	3	1
	Wrist and hand	8	2	1
	Thumb and fingers	12 (5)*	55 (36)*	8 (8)*
Lower limb	Buttock and thigh	10	3	3
	Knee and leg	15	2	1
	Ankle and foot	2	4	—
	Toes	1 (1)*	2 (2)*	—
Neck		1	—	1
Chest		2	—	—
Genitalia		2 (penis)	—	—

* Subungual tumours.

Change of temperature from warmth to cold, or less commonly the reverse, may bring on an attack, probably by inducing a change in the local circulation.

As might be expected, many patients adopt some habit or posture, or wear clothing, designed to protect the tumour from being touched.

In one case light touch would start the pain while firm pressure relieved it.[21]

The pain may be localized in the area of the tumour, but in a considerable proportion of the cases it is of a neuralgic radiating character. Pain of this type was stated to be present in 68 cases. The tumour may be found to have a particularly sensitive part ("trigger area") which must be touched in order to start the pain.

Between the attacks the patient may be free from pain or there may be a continuous dull aching in the part. Both spontaneous and provoked attacks of pain may occur in the same patient. Some patients complain only of constant aching, without distinct attacks of pain.

Glomus tumours grow exceedingly slowly and have never been known to attain a great size. Most are quite small and some measure only a few millimetres. The largest on record measured 1½ in. × 1 in.[52] In some cases it has been noted that the tumour grew to a certain size and then became stationary.

The history is usually long, often extending over many years. The longest history recorded was 43 years,[89] the shortest 2 weeks.[91] There may be a history of antecedent trauma, but this is not very common. When a glomus tumour follows trauma, it has sometimes appeared almost immediately—perhaps taking the place of an initial hæmatoma—or within quite a short time, sometimes only after a more or less lengthy interval. Twenty-nine patients gave a history of trauma, but only in 18 was there any close relation between the injury and the subsequent appearance of the tumour. Trauma has been known to cause a previously quiescent tumour to become painful.

Hyperæsthesia in the distribution of the corresponding sensory nerve has been noted, either as a temporary sequel to the attacks of pain or as a constant finding.

A glomus tumour may bleed if knocked or otherwise injured; in one case the bleeding was severe enough to require medical aid.[86]

Certain other symptoms and signs are more rarely associated with a glomus tumour. Except in 2 cases of tumour of the upper arm and of the back of the hand respectively, they have been observed only in connexion with tumours of the fingers and toes. They are:—

a. Sympathetic disturbances: Flushing[6,21,32] and warmth[6,20,21,32,52,54,71,88] or pallor[67] and coldness[49,52] of the affected part; sweating[18,21,48,52,54,71,88,89]; Horner's syndrome[6,21] (in connexion with a subungual tumour of the corresponding hand).

b. Under-development of the affected part.[6,7,19,20,32,54,70]

c. Tremor of the part.[6,7]

d. Increased tendon reflexes.[20]

e. X-ray changes: Digital, especially subungual, tumours may produce pressure-atrophy of the adjacent bone, and an X-ray picture of the terminal phalanx will then show a punched-out area of rarefaction (cupping) or diffuse loss of density.[16,18,26,32,42,53,54,57,63,70,79]

f. Changes in the nail: Subungual growths may cause the nail to become thin, ridged, irregular, brittle, or abnormally convex.[6,7,16,34,45,54,55,58,70,88,90]

Treatment.—The only adequate treatment is excision. In carrying out this small operation it should be borne in mind that the tumour is apt to be resistant to local analgesics; several authors have remarked that while the skin incision was painless the tumour itself remained sensitive. It is wise, therefore, to use plenty of the anæsthetic solution and to infiltrate a wide area on all sides of the tumour.

The tumour has been found to be insensitive to radiotherapy in 2 cases in which (in consequence of a mistake in diagnosis) this was tried.[31,66]

It is important to think of the possibility of a glomus tumour when a patient complains of severe and persistent pain which may or may not be associated with a clinically obvious tumour. A mistaken diagnosis has led on the one hand to the patient being treated, sometimes for years, as a psychoneurotic; on the other hand, to overdrastic surgery (amputation of the whole or part of a finger, which was performed in 5 cases, and other unnecessary operations, including in 1 case cervical sympathectomy).

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A CASE OF PRIMARY CARCINOMA OF THE URETER

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THE prognosis in carcinoma of the ureter is poor, and few cases survive five years after operation. The following case is recorded because the patient did survive for this period and because it was possible to follow up the course of the disease and the cause of the fatal issue.

CASE REPORT

A woman of 46 attended the urological out-patient department at Middlesex Hospital in January, 1936, giving a 10-weeks' history of hæmaturia; there had been at least one long thin clot of blood in the urine. She had some increased frequency of micturition and a little pain in the back. Both kidneys were palpable, but not tender, and the urine contained some pus cells and red blood-cells but no organisms, and was sterile on culture.

FIG. 339.—Retrograde pyelogram showing irregular filling defect in the ureter with hydro-ureter and hydronephrosis above it.

Cystoscopy showed a hyperæmic and œdematous right ureteric orifice, whilst the left appeared normal; there was no bleeding at the time.

Excretion urography revealed a small calculus in the lower end of the *right* ureter, with some dilatation of the kidney, and a gross hydronephrosis of the *left* renal pelvis. The stone was subsequently passed after cystoscopic manipulation, and retrograde pyelography was carried out on the left side. The catheter was obstructed at 15 cm., and attempts to pass it further provoked considerable bleeding. Sodium iodide injected, however, passed the obstruction, and the radiograph showed an irregular filling defect in the ureter about 2 in. long, with considerable dilatation of the ureter and renal pelvis above it (*Fig.* 339).

A diagnosis of new growth of the ureter was made with confidence.

OPERATION.—Nephro-ureterectomy was performed on Feb. 15, 1936. In the ureter was a firm fusiform mass to which the overlying peritoneum was adherent; this peritoneum was removed with the ureter. The specimen on section showed a hydronephrotic kidney

FIG. 340.—Drawing of specimen showing growth of the ureter with thickening of the wall and adherent peritoneum.

with a dilated upper ureter; below the dilatation the wall of the ureter was thickened for 2 in., and its lumen narrowed by a low papillary growth; the remainder of the ureter was normal (*Fig.* 340).

HISTOLOGICAL REPORT (Dr. R. W. Scarff, of the Bland-Sutton Institute).—"Section shows a papillary growth of the ureter with early invasion of the muscularis mucosæ by strands of transitional cells".

SUBSEQUENT PROGRESS.—The patient made a good recovery and was discharged on the twenty-fourth post-operative day. She remained well for six months, when there was a return of painless hæmaturia. Cystoscopy showed an area of growth on the right side of the bladder, mainly flat, but with some papillary projection. It was treated by perurethral diathermy. After a further six months, when there had been frequent slight attacks of hæmaturia, there was a bald tumour as large as a walnut on the left side of the bladder, with multiple small nodular seedling growths on the right side. Further cysto-diathermy was given, but the response was not very good, and it had to be repeated two months later. She now commenced to have pain in the right loin, and excretion urography showed some dilatation of the right ureter and renal pelvis; the urine remained sterile.

In July, 1937, as there was further extensive recurrence I referred her to Dr. Douglas Webster, who gave her a full course of X-ray therapy to the bladder (14,400 r.). Thereafter the recurrences were small, although they continued to appear; it was, however, possible to keep them in check by cysto-diathermy, and for one period of nine months she remained free from them.

In January, 1938, the blood-urea was 120 mg. per 100 c.c., and she was given intravenous saline with clinical improvement. She remained well until the end of the year when cysto-diathermy was again necessary. In 1939 she was treated three times and in 1940 three times.

In January, 1941, the right renal pain became worse and there was hourly frequency of micturition. Signs of cystitis began to predominate over those of growth and she was treated by continuous tidal drainage of the bladder both before and after the last diathermy in February, 1941. The urine contained much pus, but was still sterile. She became drowsy and listless, with slight jaundice and vomiting, and the blood-urea rose to 250 mg. per 100 c.c. Transient improvement followed the administration of intravenous saline, but the blood-urea rose subsequently to 290 mg. per 100 c.c. and she died from uræmia on March 19, 1941, five years and one month after the original operation.

POST-MORTEM EXAMINATION.—The right kidney was found to be pyonephrotic and its ureter dilated; there was no growth or other obstruction in the kidney or ureter, but a few soft papillomatous nodules in the bladder, which on section showed invasion by undifferentiated carcinoma.

DISCUSSION

The tendency of benign or malignant papillary growths of the urinary tract to disseminate by the implantation of seedling metastases elsewhere in the tract is well known. In this case it determined the need for cystodiathermy on thirteen occasions in five years; this repeated cystoscopic treatment probably contributed to the ultimate destruction of the remaining kidney, which had previously been damaged by a calculus. The site of some of the recurrent growth at the right ureteric orifice was also a factor in producing temporary obstruction of the sole ureter. In view of the gross pyonephrosis found at autopsy, it is remarkable that no bacterial infection was ever found in the urine on repeated examination; it may also account for the fact that until the onset of terminal uræmia she remained well enough to lead a fairly normal life and rarely stayed in hospital more than three days after each treatment.

I am indebted to Dr. R. W. Scarff for the pathological report.

PROLIFERATIVE AND OTHER LESIONS OF THE MALE BREAST

WITH NOTES ON 2 CASES OF PROLIFERATIVE MASTITIS IN STILBŒSTROL WORKERS

BY R. W. SCARFF AND C. P. SMITH

FROM THE BLAND-SUTTON INSTITUTE OF PATHOLOGY, THE MIDDLESEX HOSPITAL, LONDON, W.1

LESIONS of the breast occur in women so much more frequently than in men that proportionately less study has been devoted to the pathology of the male breast, although papers on this subject have recently been published by Horsley and Lazzari. The student of surgery is taught to recognize carcinoma and traumatic mastitis in the male, while little emphasis is laid upon the condition variously termed chronic, proliferative, or cystic mastitis, which is considered of so great importance in women, and which has been so widely discussed.

The term 'chronic mastitis' is here used to indicate any disorder of the glandular tissue of the breast which does not appear to be neoplastic, but which gives rise to symptoms and signs leading to removal by surgery. The term is admittedly bad as most of the conditions described under it both here and elsewhere are in all probability the result of endocrine disturbance, and not of infection.

We record here observations on the incidence and histology of the various types of lesion in the male breast for which mastectomy has been performed. These are compared with the findings in the female breast.

MATERIAL AND LESIONS FOUND

The material studied consists of 65 specimens from partial or total mastectomy in the male

submitted for histological examination at the Bland-Sutton Institute of Pathology, Middlesex Hospital, during the years 1924-40 inclusive.

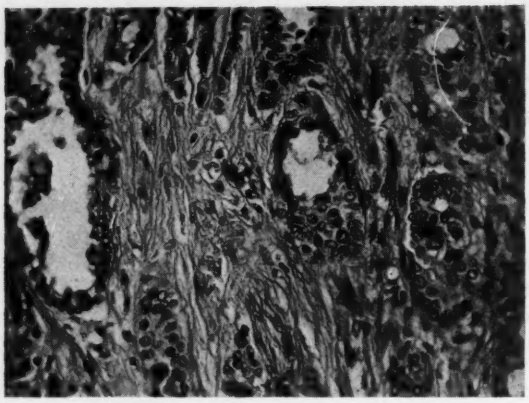

FIG. 341.—Carcinoma of male breast, Group I, with well-marked tubule formation. (× 180.)

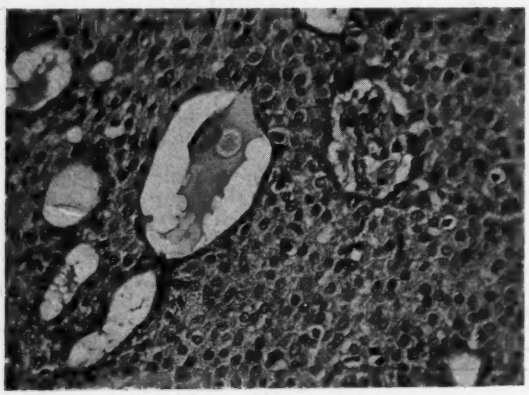

FIG. 342.—Carcinoma of male breast, Group II; spheroidal-cell carcinoma with some tubule formation. (× 198.)

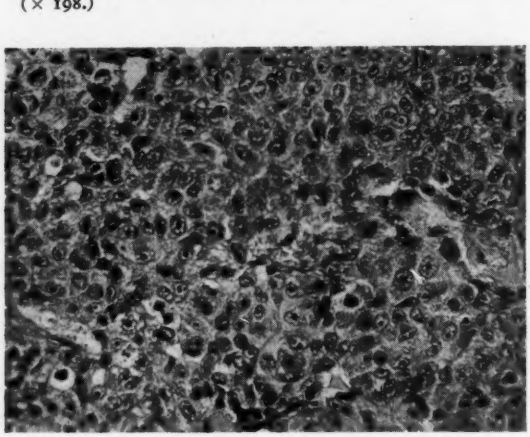

FIG. 343.—Carcinoma of male breast, Group III; spheroidal-cell carcinoma with no tubule formation, irregularity in size of cells, and evidence of rapid growth. (× 258.)

Two of these are of particular interest in that they were from workers in stilbœstrol, in each of whom hypertrophy of one breast has occurred. In addition, the findings are given of two post-mortem examinations performed on men who died of malignant disease of the breast.

In this series, there were 19 malignant neoplasms, of which 15 were carcinomata arising in the gland tissue of the breast, 3 were sarcomata, and 1 was a rodent ulcer of the nipple. There were 5 benign neoplasms, of which 4 were fibro-adenomata and 1 was a lipoma. The remaining 41 were cases of chronic mastitis.

Tumours.—Although the number is small, histological grading of the carcinomata was attempted along similar lines to those of the female breast (Patey and Scarff, 1928). Five cases were placed in Group I (the least malignant), 3 in Group II, and 7 in Group III. In making this classification it was noted that, in contrast with the female, tubule formation was present in growths in which the other factors indicated a high degree of malignancy. Typical examples of each Group are illustrated in *Figs.* 341-343.

The axillary lymphatic glands were invaded by carcinoma in 8 of our 15 cases. Freedom from glandular metastasis at the time of operation was established only in 5 of the remaining 7 cases, since with 2 specimens no lymphatic glands were submitted.

Production of 'colloid' was noted in 2 instances, both of Group I.

One of the three cases of sarcoma had a significant pathogenesis. The patient had received a bullet wound of the breast, in which a small fibroma subsequently developed. Five years later this grew larger, was removed and was seen microscopically to be a spindle-cell sarcoma. The other two sarcomata were also of the spindle-cell type; together with the single rodent ulcer of the nipple, they are included in this series for the sake of completeness, their occurrence in the breast being considered to be accidents of position, and unrelated to the pathology of the organ itself.

Of the 2 cases of malignant neoplasm which came to autopsy, one was a carcinoma of Group III, which had been treated with deep X-rays. Metastases had occurred in lymphatic glands, lungs, bones, peritoneum, and liver. The other was a spindle-cell sarcoma, for which mastectomy had been performed. Metastases were found in the lungs.

For these 19 cases of malignant disease, we were able to trace the age of appearance in all except 4. The youngest was the case of sarcoma with history of trauma, who was 34 at the time of mastectomy. Of the carcinomata, the youngest was 36 and the eldest 71, the average age of incidence being 58.

Only 4 fibro-adenomata occurred in the male breast in the period under review; 3 of them were of the pericanalicular type, the fourth being mainly intracanalicular, but containing some pericanalicular elements. The latter is shown in *Fig.* 344. The only other benign neoplasm encountered was a solitary example of lipoma.

Chronic Mastitis.—The features assessed in the pathology of chronic mastitis in the male were epithelial proliferation, formation of new fibrous tissue, and dilatation of ducts with retention of secretion. The findings are shown in *Table II*. From these figures one can state that no one pathological change occurs constantly in such severe degree as to give rise to symptoms leading to surgical intervention. In fact, 7 cases showed a severe degree of overgrowth of both epithelium and fibrous tissue, whilst these features occurred separately in the same degree in 5 and 7 cases respectively.

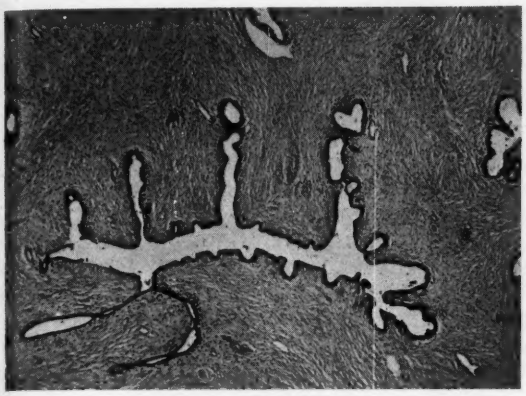

FIG. 344.—Fibro-adenoma of male breast. This field shows an area of intracanalicular type, but pericanalicular elements were present as well. (× 20.)

Table II.—HISTOLOGICAL FINDINGS IN 41 CASES OF 'CHRONIC MASTITIS' IN THE MALE

	Epithelial Proliferation	*New Fibrous-tissue Formation*	*Retention of Secretion*
None	1	5	3
Slight	11	14	23
Moderate	17	8	10
Marked	12*	14	5

* 3 of these showed "dangerous" overgrowth of epithelium.

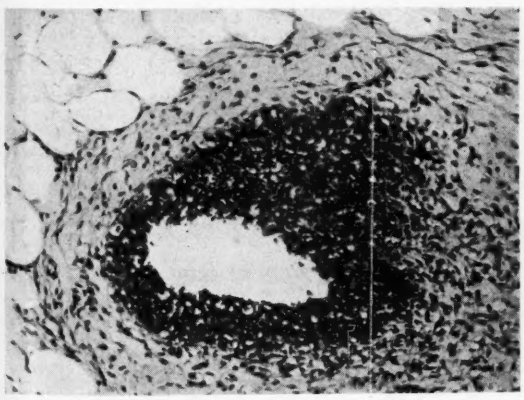

FIG. 345.—Proliferative mastitis. There is an advanced degree of epithelial proliferation with formation of new fibrous tissue (from a stilbœstrol worker). (× 114.)

In only 3 cases was there a degree of epithelial proliferation sufficiently advanced and sufficiently atypical to give rise to serious apprehension of possible malignancy. One of these is illustrated in *Fig.* 345, which also shows a moderate degree of new fibrous-tissue formation. *Fig.* 346 shows a less advanced degree of epithelial proliferation, with new fibrous-tissue formation and some retention of secretion. Marked dilatation of ducts with large quantities of retained secretion, but with little epithelial proliferation and no new fibrous-tissue formation, is illustrated in *Fig.* 347. Scanty chronic inflammatory cell infiltration was noted in only 5 cases, no instance of true chronic infective mastitis, including tuberculosis, occurring in our series. The age of the patient at the time of mastectomy was traced in 30 of the cases of chronic mastitis, the youngest being 18, and the eldest 74 years of age. In 20 cases the age was over 40, and the average was 47. The condition was bilateral in 3 cases, the longest interval between onset of symptoms in the two breasts being five years.

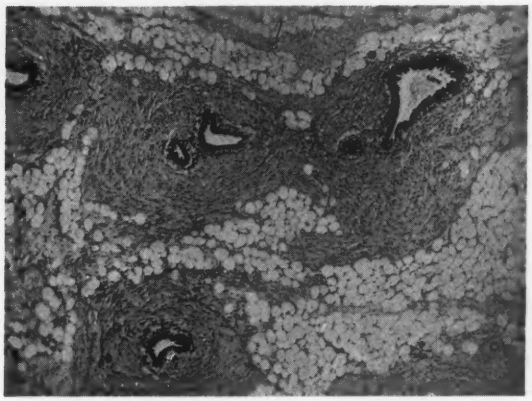

FIG. 346.—Chronic mastitis in the male, showing a moderate degree of epithelial proliferation with laying down of much new fibrous tissue around the acini. There is retention of a small quantity of secretion. (× 20.)

FIG. 347.—Chronic mastitis in the male, showing great dilatation of ducts with retention of much secretion. There is little epithelial proliferation and no new fibrous tissue. (× 20.)

Only 1 case of traumatic mastitis was noted, the diagnosis being presumed from the entire absence of proliferation of both epithelium and fibrous tissue.

BREAST CHANGES IN THE TWO STILBŒSTROL WORKERS

It is notable that only 3 of the breast sections examined and classed under chronic mastitis showed a "dangerous" degree of epithelial proliferation, and that 2 of these were from the two stilbœstrol workers to whom reference has already been made. Their ages were 18 and 23 and they had been handling stilbœstrol or its precursors for twelve and ten weeks respectively. Tenderness and enlargement of the breasts were noted three to four weeks after the commencement of this work, lasting until it was discontinued, when the symptoms gradually subsided. Biopsies were performed three to five weeks after the patients had ceased handling stilbœstrol, and the sections made revealed a marked degree of epithelial overgrowth in the acini and ducts with, in places, solid acinar formation, but no definite invasion of interstitial tissue by epithelial cells. There was a moderate degree of new fibrous-tissue formation, but little retention of secretion. These changes were more marked in the patient who had been exposed the longer time.

The only reference we can find in the literature to the effect of stilbœstrol on the human male breast is that of Dunn, who reports the clinical aspects of a gynecomastia following stilbœstrol therapy.

COMPARISON WITH LESIONS OF THE FEMALE BREAST

Table I.—Comparison of Lesions of Male and Female Breast

	Male	*Female*
Malignant Neoplasms:		
Carcinoma	15	1592
Sarcoma	3	3
Rodent ulcer	1	1
Adenocarcinoma of nipple	0	2
Squamous-cell carcinoma	0	3
	19	1601
Benign Neoplasms:		
Fibro-adenoma	4	282
Cystadenoma	0	13
Duct papilloma	0	100
Fibroma	0	12
Fibromyoma	0	1
Lipoma	1	4
	5	412
Chronic Mastitis	41	661
Various Conditions:		
Chronic abscess	0	62
Tuberculosis	0	12
Acute mastitis	0	7
Simple cysts	0	64
Fat necrosis	0	26
	65	2845

During the years 1924–40, material from 2845 mastectomies performed on women was examined histologically at the Bland-Sutton Institute. The distribution of benign and malignant lesions in this series is shown in *Table I.*

It will be apparent from a study of these figures that the commonest lesion in the male to cause symptoms requiring operation is chronic mastitis, which occurs approximately in the proportion of two cases to every one of carcinoma. In women, the proportion is reversed, cancer being more than twice as common a finding at operation as chronic mastitis.

A detailed study of chronic mastitis in women does not come within the scope of this work, but for purposes of comparison with the male series, the 661 female cases were classified along similar lines. It was found that there was a "dangerous" degree of epithelial proliferation in 103, a moderate or marked degree in 243, and that 315 showed fibrous or cystic changes only with little or no epithelial proliferation. That in general chronic mastitis is less serious in the male than in the female breast is shown by comparison of the incidence in the two sexes of "dangerous" epithelial overgrowth in proliferative mastitis. Thus of the 661 cases of this condition in the female, a "dangerous" degree of overgrowth was noted in 103, or roughly 1–6, whereas in the 39 male cases a similar state of the epithelium was remarked in only three instances, the proportion being 1–13, or roughly half that of the female series.

SUMMARY AND CONCLUSIONS

Material removed surgically from the male breast and submitted for histological examination over a period of seventeen years was analysed and compared with similar lesions occurring in women during the same period. Chronic mastitis occurred in the male twice as commonly as cancer, the reverse proportions obtaining in women, but a degree of epithelial proliferation sufficiently advanced to give rise to apprehension of possible malignancy occurred in cases of chronic mastitis in men only half as frequently as in women.

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AN OPERATION FOR INCREASING THE RANGE OF INDEPENDENT EXTENSION OF THE RING FINGER FOR PIANISTS

By C. G. BATTY-SMITH

FROM THE PUBLIC HEALTH LABORATORY, WAKEFIELD, YORKSHIRE

Ever since man invented keyed instruments and began to play upon them, generations of students have been worried and disheartened by the weakness and limited range of extension of the ring finger. Generations of professors have written volumes of exercises designed to induce this finger to move independently while those on either side of it were held down and the hours of drudgery at these exercises has, until recent years, occupied a prominent place in piano teaching. That the practice of such exercises was largely fruitless is proved by their employment by enlightened teachers to-day to a very much smaller extent than formerly and in a less savage form.

The lift of the ring finger, with the hand placed upon a flat surface in the piano-playing position, varies from about $\frac{1}{8}$ in. in the worst cases to about $\frac{5}{8}$ in. in the most fortunate, and the writer's two hands represented these two extremes, the right hand having the disability. If the right little finger was flexed upon the palm, the ring finger was compelled to follow nearly all the way; in the left hand the little finger could be so flexed and the ring finger was only forced to leave the position of full extension by about 25° to 30°.

The question may be asked, "Have all accomplished pianists a high degree of independent extension in their ring fingers?" An investigation of this subject would certainly be interesting, but the writer has not been in a position to undertake it. Two good pianists known to him have a remarkable degree of freedom, and two others (one of them a first-rate concert pianist) suffers from the disability in an extreme degree. It may be argued, therefore, that if a high degree of technical facility can be obtained in the presence of the disability it cannot be of real importance. The criticism is justified and demands a reply.

A good pianist in playing keeps his finger-tips very close to the keys; he does not raise them high to give a hammer-blow for incessant practice has developed an extraordinary power and responsiveness in his flexor muscles (the lumbricals are said to play the greatest part) which enables him to produce the necessary force from key-level. The natural power of flexion of the ring finger is rather less than that of the others, but there is as a rule no serious lack of differentiation in the flexor group of muscles such as is found on the extensor side. This being so, it is open to the pianist by constant practice to acquire an adequate responsiveness and flexor power in his ring-finger muscles.

Besides this, various devices are employed to bolster up the power of the weaker fingers. One school of pianistic thought has advocated the use of the weight of hand and fore-arm to supplement muscular power and another makes use of forearm rotation to achieve the same purpose. It is easy to see that, as the ring finger lies slightly to one side of the long axis of the forearm a key can be struck with considerable force by forearm rotation without bringing the flexor muscle into play at all. Power to strike a key can also be obtained by pushing the finger towards the key with the forearm in certain situations.

It is thus clear that the handicap of the weak finger can be overcome by the pianist by certain forms of 'trickery' which doubtless have been instinctively employed by all 'gifted' students since long before the theorists ever set them down on paper. In addition, a ripe experience is of as much value to the pianist as it is to any other skilled worker. During a discussion on the ring-finger difficulty, a fine pianist once said to the writer, "If I cannot use that finger for a passage, I use another one". What in fact he meant was that an accomplished player has the knowledge and experience to devise an alternative way of playing a difficult passage and the dexterity to carry it out.

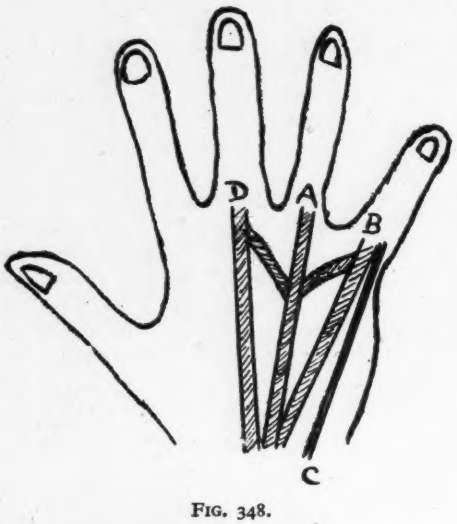
An apology is due for the fact that this discussion has strayed so far from the surgical field, but the proposed operation must be justified and the justification will now be attempted.

The situation amounts to this, that when through years of unremitting practice the whole armoury of muscular development, technical experience, and pianistic cunning have been acquired, the actual limitation of extension of the ring finger may matter little, but that to those less gifted and to those beginners who suffer from a severe degree of disability the road to clear and rapid playing would be shortened by the proposed operation. The psychological effect produced by the disappearance of a difficulty and the resulting optimism could not but be of great advantage to both student and teacher. Finally, the writer can say from having undergone the operation that it has been an unqualified success. Many passages that formerly defied all efforts to play them clearly have become easy, and there has been no disadvantage of any kind observed in the everyday use of the hand during the eight months since the operation was performed.

THE RATIONALE OF THE OPERATION

It has generally been supposed that the limited range of extension of the ring finger is due to the presence of two tendinous bands running from the tendon of the extensor digitorum communis associated with the ring finger to the tendons of the middle and little fingers. These bands have usually been illustrated in text-books of anatomy as running in the directions shown in *Fig.* 348. Were this arrangement in fact present, a kind of 'valvular' effect would be produced, for while, owing to the direction of the bands, a pull on tendon A could not extend the ring finger without also extending the middle and ring fingers, a pull on tendons B and D would extend the respective fingers and the bands would merely loosen without extension of the ring finger occurring. So long as this portrayal of the arrangement of the bands was a true one it was a practically perfect explanation of the observed facts, but examination of even a few specimens shows that the truth is not so convenient.

In the writer's right hand the arrangement was as in *Fig.* 349, the bands being square plates rigidly joining the three tendons and certainly quite incapable of allowing any 'valvular' effect such as that just described.



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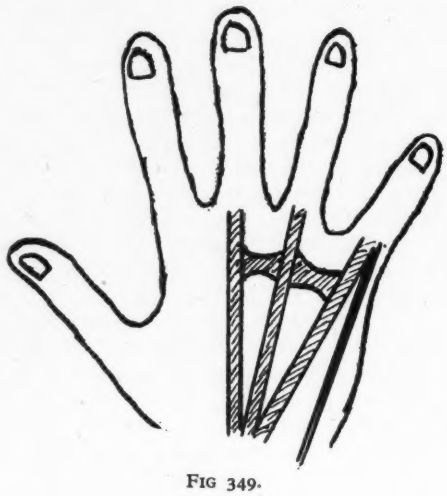

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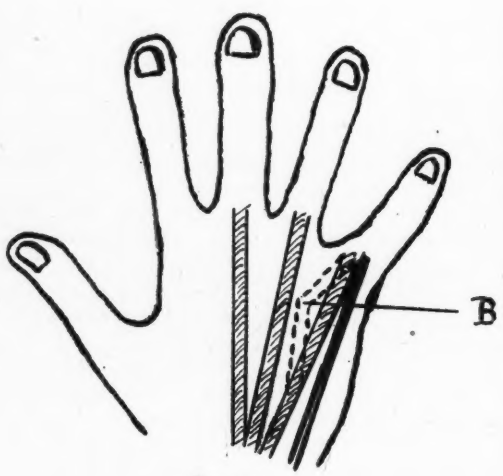

FIG. 351.

FIGS. 348–351.—Arrangement of extensor tendons (diagrammatic). A, B, and D, Tendons of extensor communis; C, Double tendon of extensor digiti quinti proprius. For significance of B¹ in *Fig.* 351 *see* text.

In another specimen examined the arrangement was as in *Fig.* 350, in which there could hardly be said to be two separate tendons but rather a fairly wide sheet of tendon containing fenestrations.

In other specimens a multiplicity of arrangements of bands and small strands of tendon may be observed, and though the writer has examined but few, the classical arrangement does not seem common.

The First Operation on the Author's Hand.—In accordance with the generally accepted

views, the writer had the interfering bands completely removed by Mr. W. McKissock. The results were extremely disappointing as not a trace of benefit resulted. It was at first hoped that the desired freedom might develop after the elapse of time and with judicious exercise, but as after five years there was not the slightest improvement the writer again became interested in the subject.

It was concluded that the most probable explanation was that the muscle bellies of the extensor communis associated with the ring and little fingers were not physiologically differentiated, but were acting as one muscle which was 'ring-finger conscious'. In consequence of this, the ring finger would not be able to be extended if the little finger was flexed, the flexors being more powerful than the extensors. Separate extension of the little finger was, as usual, possible and it was thought that this movement must be performed entirely by the extensor digiti quinti proprius. It was therefore decided to operate a second time and to extirpate the tendon of the extensor communis to the little finger. The writer thereupon consulted Mr. Philip Allison, who agreed that the rationale of the operation was sound and thought it had every prospect of success.

The Second Operation.—A local anæsthetic was used as the patient's co-operation was essential. The incision, proposed by Mr. Allison, began over the proximal end of the first phalanx of the ring finger, curved outwards to the level of the outer side of the fifth metacarpal, and in again to a point between and level with the bases of the fourth and fifth metacarpals. The flap so formed was turned up and the tendon of the extensor digiti quinti proprius identified. (This muscle is absent in some subjects.) The field of operation being fully exposed, the behaviour of the tendons was observed during extension of the ring and little fingers (*Fig.* 351).

Direct observation completely confirmed the author's hypothesis as to what was taking place. Extension of the ring finger caused tightening of tendon B while extension of the little finger was evidently performed entirely by tendon C (extensor digiti quinti proprius), for when this was done tendon B became quite slack and took up position B^1 as shown by the dotted lines. Accordingly, between 1 in. and 1½ in. of tendon B were excised and the range of extension of the ring finger was immediately increased. The wound was closed with a continuous mattress suture. On the sixth day the stitches were removed.

It should be remembered that the bands had been removed on a previous occasion, but it would be advisable to remove the band between the tendons to the middle and ring fingers when performing this operation so as to leave no possibility of restriction from this cause.

After-treatment and Results.—The fingers were occasionally moved during the week following operation to prevent the tendons from sticking, but no serious exercise was undertaken until the twelfth day. Simple exercises were then performed on three occasions during each day and consisted in: (1) Holding the little finger lightly on to the palm while extending the ring finger as strongly as possible twelve times; and (2) With all the fingers fully extended, flexing the little finger as rapidly as possible on to the palm twelve times while keeping the ring finger as fully extended as possible.

At first both these exercises were difficult to perform, but improvement was surprisingly rapid, and after persisting with them regularly for three weeks no further improvement was noticed. After this a first trial at the keyboard was made, when the benefits of the operation were immediately evident. Although no exercises were performed beyond three weeks from the operation and very little keyboard practice has been done, it is noticeable that now (eight months later) there is a further improvement in the independence of the finger, which can be lifted about ⅝ in. with the hand in the piano-playing position and can be extended to within about 25° to 30° of complete extension while the little finger is held flexed on the palm.

DISCUSSION

It is obviously dangerous to draw conclusions from a single case, but the complete failure of the first operation does suggest that the tendinous slips are not a factor in the ring-finger disability. It would be expected that if the muscle bellies of the extensor communis for the ring and little fingers were well differentiated, any tendinous bands would become sufficiently stretched while they were pliable in childhood so that they could not interfere with independent extension. If, on the other hand, the bellies were acting as a single muscle there would be a tendency to form rigid and inelastic bands. It is suggested, in other words, that it is the state of differentiation of the muscle which is responsible for the nature and behaviour of the tendinous bands and not vice versa. It is interesting that, since the operation, the writer's right ring finger has acquired a degree of freedom almost identical with that naturally possessed by the left ring finger, which again suggests that the tendon slips in the left hand (which are certainly present since they are visible beneath the skin) are not handicapping movement to any great extent.

CONCLUSIONS

The writer concludes from personal experience that this operation is well worth undergoing by any piano or organ student who is seriously handicapped in his studies by the possession of a ring finger with very restricted extension. The curved incision described has healed extremely well and will before long be practically invisible.

It would, perhaps, be difficult for people with the artistic temperament to persuade themselves

to undergo such an operation, partly from fear of the procedure itself, but partly also from a vague fear that the hand might be permanently ruined, though if even a small number of students were known to have undergone the operation with successful results, no doubt others would follow.

SUMMARY

A successful operation is described for increasing the range of independent extension of the ring finger for pianists. The rationale of the operation and the pianistic considerations making it desirable are discussed. The lift of the ring finger with the hand in the piano-playing position was increased in the author's hand from about $\frac{1}{8}$ in. to $\frac{5}{8}$ in.

My thanks are due to Professor Durward, Professor of Anatomy at the University of Leeds, for his help with the anatomy of the part and for showing me several preserved specimens; to Mr. Philip Allison, Assistant Surgeon to the General Infirmary, Leeds, who entered thoroughly into the spirit of this small experiment and spared no pains to secure a successful result; and also to Mr. Wylie McKissock, Surgeon to the Maida Vale Hospital, who performed the first operation which was unsuccessful through no fault of his.

MUCOID ADENOCARCINOMA OF THE URINARY BLADDER

By H. SPENCER

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Rare and unusual types of pathological lesions seldom fail to attract an undue share of attention, but a recent case seen by the author stimulated a search of the surgical literature for descriptions of mucoid carcinoma of the urinary bladder.

The recognition of more than one type of carcinoma of the bladder appears to have originated within the last thirty years, but since the description given by Ehrlich in 1901 of an adenocarcinoma in an ectopic bladder, at least 28 cases have been recorded, mostly in the American literature. Begg, in an exhaustive survey in 1931, collected 44 cases of what he has described as tumours of the urachal remains, of which 19 were found to be mucoid carcinomata. De Waard (1939) has described a further 2 cases, Lowrey (1939) 5 cases, McCown (1940) 1 case, Connell (1940) 1 case, and several examples of benign adenomata have occurred. The report of the American Urological Association's Registry of bladder tumours quotes an incidence of the type of growth under discussion as 1·8 per cent of the total of 923 bladder carcinomata (Hamm, 1940). St. Michael's Hospital, Toronto, have reported a figure of 5 adenocarcinomata out of a total of 300 cases of bladder carcinomata (Lowrey, 1939), and the Mayo Clinic 5 cases among 333 bladder tumours over a period of ten years (Scholl, 1922).

CASE REPORT

The present case was first seen at St. Mary's Hospital in February, 1938. G. A. B., a young man of 23, attended the hospital with a two-months' history of hæmaturia, worse at the end of micturition. He also complained that he thought the act incomplete and that subsequent straining to complete the emptying of the bladder caused him pain. He had no diurnal or nocturnal frequency.

A radiographic examination of the bladder area showed a ring-shaped calcified opacity (*Fig.* 352). This was erroneously considered to be due to a large calcium deposit in a vesical calculus.

Cystoscopic examination showed an encrusted tumour on the right anterior wall of the bladder, which when poked gently with a ureteric catheter appeared completely fixed.

Operation.—Early in May, 1938, a 3-in. suprapubic, midline incision was made and the bladder exposed. A swelling "the size of a walnut" was found at the apex of the bladder subjacent to the anterior abdominal wall (*Fig.* 353). This swelling was caused by a smooth, immovable tumour with no surrounding inflammatory reaction. The tumour was excised and the bladder wall sutured.

Fig. 352.—A radiograph of the pelvis, showing a ring-shaped calcified opacity in the vicinity of the bladder.

Subsequent Progress.—Convalescence was uneventful except for an attack of tonsillitis. The patient was re-examined in May, 1939, at a follow-up clinic and was found to be symptomless and there was no apparent recurrence of the growth. In September, 1940, two and a quarter years after his

operation, he complained that his right leg began to ache and later he developed dysuria with pain referred to the urethra after micturition. Following this his urine was blood-stained from time to time, and he suffered from diurnal frequency.

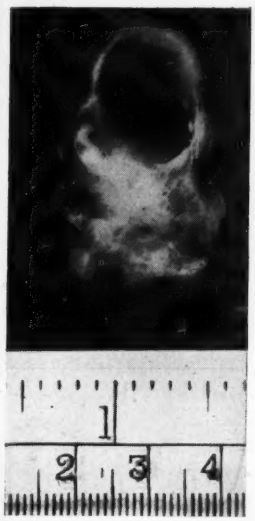

FIG. 353.—A photograph of the complete specimen removed at the first operation, showing the cyst above and the growth below.

In May, 1941, he was admitted to Park Prewett Hospital, Basingstoke, and was found to have a hard, fixed, suprapubic mass immediately behind the anterior abdominal wall. A cystoscopic examination showed two nodular growths in the vesical wall, one situated at the base behind the trigone and the other in the dome of the bladder.

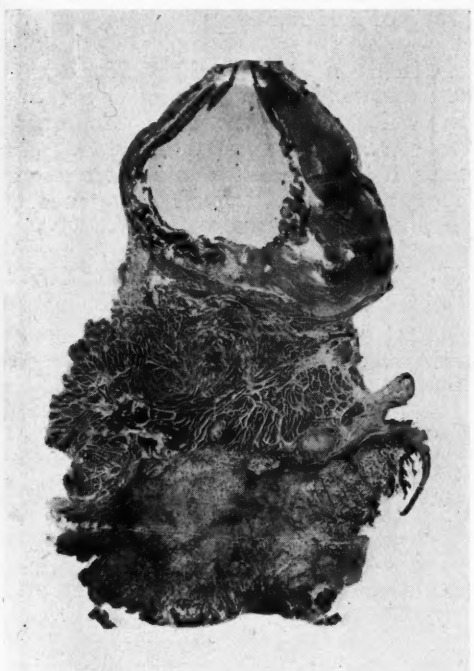

FIG. 354.—A low-power view of the complete section, showing the urachal cyst above, the growth below, and in the centre the muscular layer of the bladder infiltrated with masses of growth. Normal bladder mucosa may be seen spreading over both edges of the growth. (× 2·1 approx.)

SECOND OPERATION.—A week later a midline, suprapubic incision was made and the direct spread of the growth into the perivesical tissues was found. The growth had spread too far for operative treatment and the wound was closed. During convalescence an X-ray was taken of the pelvic bones, but it showed no bone metastases. When the patient was discharged from hospital he was still complaining of hæmaturia and of the pain radiating down his right leg, for which he was receiving analgesic drugs.

PATHOLOGICAL REPORTS.—The report on the specimen obtained at the first operation was as follows: "Macroscopically, an irregular mass 4·0 × 2·2 × 3·8 cm., with an ulcerated convex surface 3·0 × 2·5 cm. at its broader end, bordered by œdematous vesical mucosa. Immediately beneath the ulcerated mucosa a number of translucent areas separated by a network of fibrous tissue. The muscle layer is also broken up by bands of fibrous tissue and mucus. At the lower pole of the specimen outside the muscularis is a cyst 1·8 × 1·4 cm., filled with mucus and with calcified areas in the periphery".

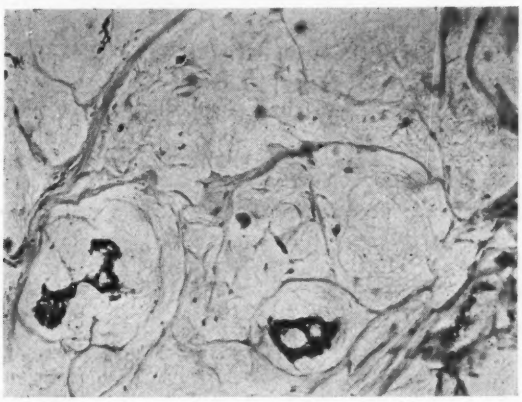

FIG. 355.—Microphotograph of the growth, showing islets of carcinoma cells surrounded with mucus. (× 76.)

The microscopical report on the sections stated: "A portion of bladder wall with a cyst formed by fibrous tissue and containing mucoid material and a little calcium. There is a columnar-celled mucoid carcinoma at the edge of the cyst infiltrating the muscular wall. Mitotic figures are numerous" (*Fig.* 354). The microscopical report on the section obtained at the second operation in May, 1941, was as follows: "The tissue submitted for section is invaded by a mucoid adenocarcinoma. There are isolated islets of carcinoma cells with a good fibrous-tissue reaction and mitotic figures are not numerous" (*Fig.* 355).

DISCUSSION

The salient clinical features of the cases described in detail in the literature are similar to those of the transitional-celled type of growth which is the more usual form found in the bladder, except that disorder of bladder function may be noticed earlier in the disease. The symptom which occurs in most cases is hæmaturia, but other less common symptoms are frequency, dysuria, and suprapubic pain. In most of the described cases the patients were males, the proportion being about 4 to 1. The average

age was 47, and a few of the cases were associated with ectopia vesicæ.

Pathologically, adenocarcinomata of the bladder appear to be divided into distinct groups, those occurring primarily in the apex of the organ and those occurring in the region of the trigone and the posterior wall.

Much discussion has arisen as to the possible origin of the columnar-celled carcinoma of the bladder. At one stage in the development of the fœtus the urogenital sinus and the posterior portion of the hind-gut form a single outlet or cloaca. Subsequently the cloacal septum grows down from above to divide the cloaca into the future urinary and alimentary portions, represented in the adult by the urinary bladder and rectum respectively. The rectal side of the septum develops glandular structures which are the precursors of the adult intestinal mucosa of the rectum; and it has been suggested that islets of such glandular tissues find their way on to the vesical surface of the septum and come to occupy a position on the posterior wall of the bladder. This developmental theory associated with the persistence of the fœtal state into adult life was thought by Enderlen to explain the origin of the growths from the trigone and posterior wall of the bladder. It was thought by the same author to be unnecessary to postulate the persistence of the columnar-celled epithelium into adult life, as it only required a certain slight stimulus to convert the normal bladder epithelium by a metaplastic change into the columnar state. Such a stimulus may be provided by inflammation or ectopia vesicæ. Also the absence of glandular tissue in the bladder wall of the embryo was claimed by Lendorf. The same author showed that in children the formation of solid buds of columnar cells, occurred, and that in later life these were capable of forming definite glands. When inflammatory changes were present the glands secreted mucus and the condition of cystitis cystica resulted. It is possible to demonstrate the formation of adenomata from the multiple tumours present in the condition of cystitis cystica.

Buckel, quoted by De Waard (1939), has suggested the para-urethral glands of Albarran at the neck of the bladder to be the origin of the basal type of growth.

The commoner locality to find columnar-celled carcinoma of the bladder is at the fundus of the viscus extending upward towards the obliterated part of the urachus (Ferrier, 1936), and the case described above is probably one of this group. Adami, quoted by De Korte (1918), and many others have suggested that the urachal remains give rise to this type of growth. Begg (1931), in his review of the subject, has shown that neoplasms may arise from the urachus anywhere between the bladder and the umbilicus, and that simple retention cysts formed from unobliterated portions of the fœtal structures are commonly found at post-mortem examinations. Suchannek (1879), in a routine study of many specimens of the normal obliterated urachus, has also demonstrated the presence of goblet cells and mucin in a few specimens. It is thought that islets of these cells are those responsible for the formation of mucoid adenocarcinomata in the fundus of the bladder of the adult.

It is suggested that the growth in the above-described case arose from islets of glandular cells along the course of the obliterated urachal canal where this structure traversed the apex of the bladder wall. The cyst immediately above the growth is probably a simple retention cyst of unobliterated urachus, in which calcification has occurred.

SUMMARY

A case of mucoid adenocarcinoma of the bladder has been described and a résumé of the literature on similar cases has been presented. The prevailing embryological views as to the aetiology of the condition and the apparent clinical division into apical and basal growths has been described. Reasons have been advanced for the classification of the present case amongst the group of apical growths.

In conclusion I wish to thank Professor C. A. Pannett for the use of his clinical notes; Dr. Rohan Williams for the X-ray report; and Professor W. D. Newcomb and Dr. G. E. Harre for their helpful criticism and interest. I am also indebted to Mr. Pearce for the preparation of the photomicrographs.

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FRACTURES OF THE OLECRANON PROCESS

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A SURVEY of recent literature reveals a considerable divergence of views on the treatment of fractures of the olecranon. The following methods of treatment all have their advocates :—

Non-operative.—

1. No immobilization and the early resumption of physiotherapy and active exercises.

2. Closed reduction of the fracture and immobilization until bony union is obtained.

Operative.—

1. Immediate or delayed open reduction of the fracture and accurate fixation of the fragment to the shaft by : (*a*) Non-absorbable material—e.g., wire, plates, screws, and Steinman pin ; (*b*) Absorbable material—e.g., catgut, kangaroo tendon, fascia lata, fascial strips from the triceps expansion.

2. Excision of the detached fragment of the olecranon and repair of the triceps expansion.

The history of the treatment of this fracture has been well reviewed by Ellsworth Eliot (1934).[1] Before the days of aseptic surgery these fractures were usually immobilized in full extension for from four to six weeks. Because of the danger of a stiff elbow with permanent loss of flexion, the position of mid-flexion was adopted. This, however, almost invariably resulted in non-union of the fracture owing to the wide separation of the fragments.

In 1884 Lister performed the first open reduction of a fractured olecranon and this method of treatment rapidly gained popularity. In 1894 Sachs reported excellent results with a rapid restoration of full function by dispensing with any form of splinting, allowing the arm to hang down by the side and instituting early massage. At the end of two weeks active movements were started and full function was said to be regained in six weeks. This was prior to the days of X rays and undoubtedly in most of these cases only fibrous union was obtained.

It was pointed out by Perkins (1936)[2] that although good function is possible when union occurs by fibrous tissue, two disabilities invariably result : the triceps muscle becomes weaker because when the muscle is at rest the distance between its origin and insertion is shortened and when it contracts part of its energy is used in taking up slack. The second disability is that as a result of the upward displacement of the olecranon process there is no hollow on the back of the humerus to receive the beak of the olecranon. As the elbow is not a weight-bearing joint these disabilities are much less important than similar disabilities in the knee-joint, where a strong quadriceps and the ability to extend the knee-joint fully are of importance in the maintenance of the erect posture.

Daland (1933)[3] reviewed 48 cases of fracture with 24 end-results. His conclusions were that closed reduction was very unsatisfactory and that open operation is invariably indicated where any separation exists. There appears to be general agreement that the best suture material to employ is fascia lata or fascial strips from the triceps tendon, as suggested by Rombold (1934).[4]

There is a considerable divergence of views as to when to start movements following operative fixation. Perkins states that the elbow should be immobilized in plaster at a right angle and the arm bandaged to the side, to prevent triceps contractions, for fourteen days. On the other hand, Daland commences active movements three or four days after operation.

Watson-Jones (1940)[5] considers that reduction by closed manipulation can often be achieved by full extension of the elbow and firm pressure over the fragment. Conservative treatment is only justified if apposition of the fragments is accurate, and immobilization must be continued for five weeks. Recovery of full flexion may be delayed for a year and frequently requires gentle manipulation at intervals. He reserves treatment by excision of the fragment for elderly people, especially when the detached fragment is small.

Naughton Dunn (1939) expressed the opinion that fractures of the olecranon process treated on accepted lines are, in the hands of the most experienced surgeons, always an anxiety. We are dealing with a disturbance of the continuity of an articular surface necessitating the maintenance of accurate apposition of the fragments until bony union is firm. Movements, even when fixation by non-absorbable material is employed, can seldom be allowed under six weeks if the risk of tilting of the fragment is to be avoided. In common with other fractures involving a joint surface, restoration of full movements is delayed and often incomplete, and a painful arthritis a not uncommon sequela. It was in order to avoid these complications that he advocated the treatment of these fractures by removing the detached fragment and re-attaching the triceps expansion to the upper end of the shaft of the ulna.

The exact type of treatment employed varies with the age of the patient, the type of fracture sustained, and the whim of the surgeon in charge of the case ; but, roughly, the position to-day is that closed reduction, although frequently tried, is rarely successful and is reserved for the younger patients. Open reduction and some

form of internal fixation is used for the majority of patients, and a sling and the early resumption of active movements—although this invariably gives rise to non-union—is accepted as the best treatment for the elderly patient in whom an operation is contra-indicated. There remains a small group of older patients with small detached fragments for which excision of the olecranon process is reserved.

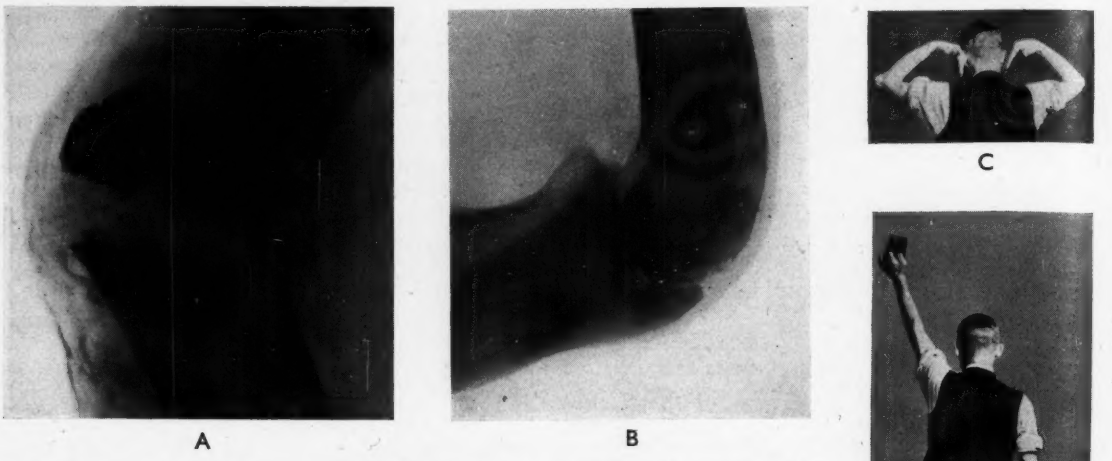

FIG. 356.—Mr. C., aged 63. A, Pre-operative radiograph; B, Post-operative radiograph; C, Photograph showing the degree of flexion 3 months after operation; D, Showing the power and degree of extension, patient holding a 12-lb. weight.

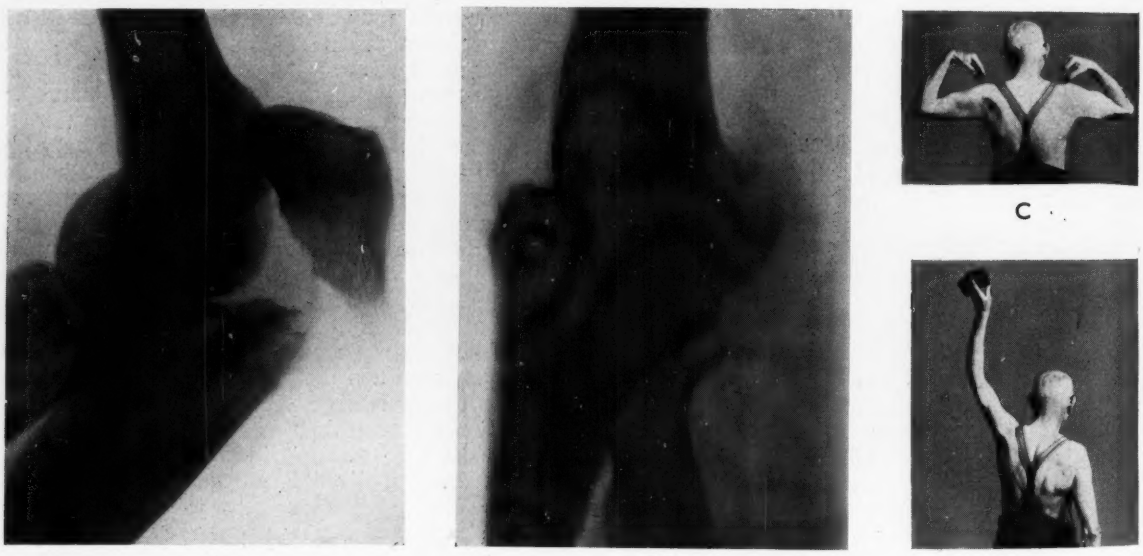

FIG. 357.—Mr. B., aged 65. A, Pre-operative radiograph; B, Post-operative radiograph; C, D, Photographs taken 6 weeks after operation, showing (C) range of flexion, and (D) slight limitation of extension, which has since disappeared—the patient is holding a 12-lb. weight.

PERSONAL CASES

The cases which form the subject of this review have been under the care of the late Mr. Mitchell Smith and myself at the North Staffordshire Orthopædic Hospital and various associated hospitals during the past eight years. From a study of the earlier of these results the following conclusions were reached:—

1. That a satisfactory reposition is very seldom obtained by closed reduction and that the method requires prolonged fixation with the elbow in extension and should be attempted only in the young. Incidentally, a position of a few degrees' flexion instead of complete extension is much more likely to produce accurate reduction.

2. That while a useful range of movement can be obtained by using a sling and instituting active exercises, the weakness of the triceps, the painful loose fragment, and the inability to extend

the arm fully did not justify the use of this method except in the elderly where any operative treatment is contra-indicated.

3. That while perfect results can be obtained by open reduction and fixation by absorbable material in younger patients, arthritic changes and permanent stiffness are frequent enough to make us ask ourselves whether any other method can produce better results. In addition, convalescence is always prolonged and it is infrequent to find a heavy worker return to his employment in less than six months.

exhibited any lateral instability of the elbow-joint and in this case the fracture involved the coronoid process, and the plateau for the reception of the lower end of the humerus when the elbow was extended was completely broken up.

The stability of the elbow-joint, as shown by experiments on the cadaver, does not appear to depend on the presence of the olecranon process in the olecranon fossa, but on the integrity of the upper surface of the coronoid process and the lateral ligaments of the elbow-joint, together

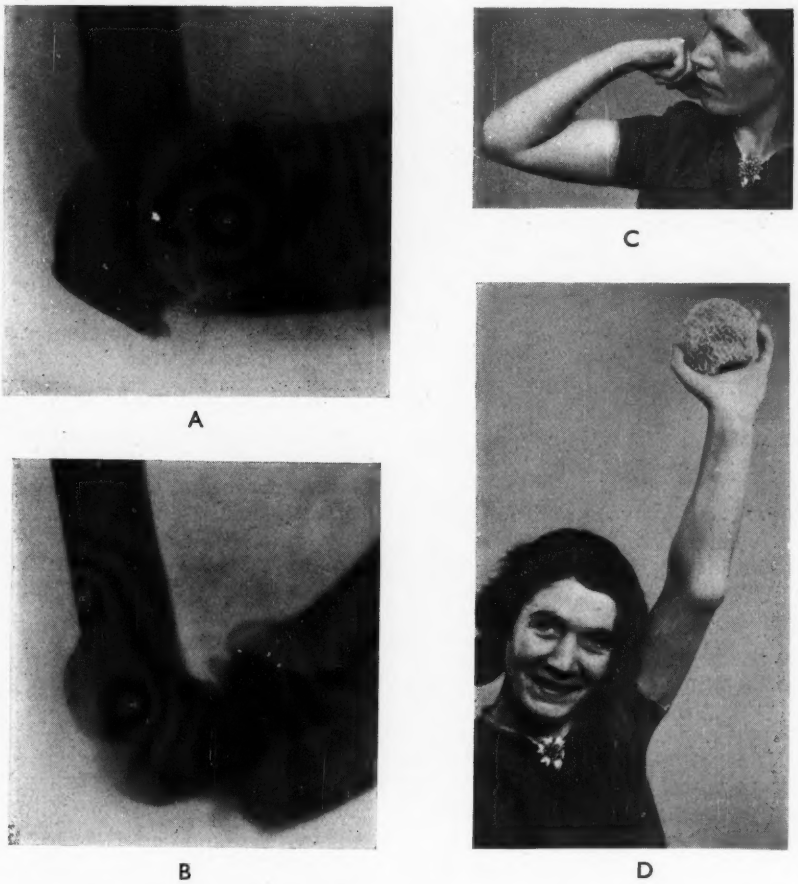

FIG. 358.—Mrs. H., aged 46. A, Pre-operative radiograph; B, Post-operative radiograph; C, D, Photographs taken 4 months after operation, showing (C) degree of flexion, and (D) power and degree of extension—patient is holding a 10-lb. weight.

REMOVAL OF DETACHED FRAGMENT AND TRICEPS REPAIR

Objections that have been raised to this type of treatment are that, whilst it is satisfactory for the elderly patient with a small detached fragment, (*a*) removal of large portions of the olecranon process gives rise to lateral instability of the joint; (*b*) the power of the triceps muscle is impaired; (*c*) the last few degrees of extension of the elbow are not obtained; and (*d*) the point of the elbow is lost.

In this series of cases only one patient with the control of the biceps and triceps muscles when their tone is restored.

None of these cases exhibited impairment of the power of the triceps muscle as estimated by a spring balance at least six months after the injury. Two or three of the earlier cases had exhibited a slight limitation of full voluntary extension of the elbow-joint, but this has since been avoided by suturing the triceps expansion more firmly to the upper end of the ulna and immobilizing the elbow-joint in complete extension for eight to ten days. There is no objection to the operation on cosmetic grounds.

The advantages of this type of treatment are: (1) A more rapid restoration of movement and return to work; (2) There is no possibility of arthritic changes developing and no danger of non-union or mal-union of the fracture; and (3) The ease and rapidity of the operation.

Operative Note.—In addition to the firm suturing of the triceps expansion and immobilization of the elbow in full extension, the only other important points are that the fragment should be removed by a subperiosteal dissection and that the projecting sharp angle of the upper end of the ulna should be smoothed off.

In the after-treatment reliance is placed entirely on supervised active movement and no forced movements are permitted. This type of treatment is advocated: (1) for all patients approaching middle age where the fracture is above the coronoid process; (2) as an alternative operation for younger patients where early return to work is important; (3) in every case of badly comminuted fracture where the articular surface is broken up and where any difficulty is experienced in apposing the fragments or maintaining them in position.

RESULTS

	Cases
A. Open reduction and fixation	14
B. Closed reduction and fixation	3
C. No fixation	3
	20
D. Treated by excision of olecranon process and repair of triceps	20
	40

A. **Open Reduction** (14 cases).—Absorbable material only used (catgut, 12; fascia lata, 2).

Fixation: 6 weeks' immobilization in plaster, with elbow at a right angle.

Results:

6 cases under 35 years, complete recovery.

8 cases over 35 years, incomplete recovery.

Analysis of 8 cases with Incomplete Recovery.—

3 cases (average age 65 years)—Marked arthritis with pain and limitation of movement.

5 cases (average age 50 years)—Permanent limitation of movement; no pain.

3 patients unable to resume work and all had prolonged convalescence.

B. **Closed Reduction and Fixation** (3 cases).—

Ages: 2 cases under 20 years; 1 case, 55 years.

Results:

Under 20: 1 case, full recovery; 1 case, limited extension.

55 years: 1 case, flexion and extension limited.

C. **Sling and Early Movements** (3 cases—average age 75 years).—

Results:

1. Some limitation of full extension
2. Triceps weak
3. Tenderness over loose fragment

} present in every case.

D. **Excision of Olecranon and Triceps Repair** (20 cases).—

Average age: 54 years (oldest 75 years; youngest 30 years).

Size of fragment removed:

Less than half olecranon, 12;

More than half olecranon, 8.

Results (*Figs.* 356–358): 4 cases have been under observation for three years and all have continued with their original work without symptoms. The remainder have been under observation for periods varying from six months to two years.

One case, which was associated with a fracture of the coronoid process and the head of the radius, has some lateral instability of the joint and impairment of the power of the triceps muscle.

Two cases exhibit slight permanent limitation of full extension of the joint. The power of extension, however, is not impaired and these patients perform their original occupation without any discomfort.

The remaining 17 patients have a full range of movement without any symptoms or impairment of function.

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THE SEMILUNAR CARTILAGES

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DEVELOPMENT

THE future lower limbs can be recognized as early as the second month of intra-uterine life as small nodules projecting from the sides of the fœtal body. Each bud consists of a mass of undifferentiated mesenchymatous tissue, the cells of which are small, round, with large nuclei and comparatively little protoplasm. Soon the mesenchymatous cells undergo an alteration into chondrogenous tissue by an increase of the protoplasmic-cell content, the nuclei meanwhile remaining unaltered. For a brief period the limb bud consists of this mass of chondrogenous tissue, which is further altered by the deposit of cartilaginous and intercellular material in isolated islands. These areas steadily increase in size and approach each other from above and below, leaving a narrow area of unaltered chondrogenous tissue from which the joint structures develop. This undifferentiated mass, or intermediary disc, splits at a later stage, its upper and lower layers fusing with the adjacent cartilaginous ends of the femur and tibia to form for them a perichondral covering, the gap left by the splitting forming the future joint space. The development of the joint cavity is a gradual process, at first in its upper and anterior section, later continuing backwards and inwards until the separation has been completed. A similar process occurs later on the distal side of the disc, between it and the upper end of the tibia. This second splitting takes place during the latter part of the fourth month, when a vertical septum, derived from the same mesenchymatous tissue, grows backwards through the middle of the joint from the posterior aspect of the patella. The joint cavity consists then of four separate cavities which remain distinct for a short period; the two original sub-divisions are restored by the partial absorption of the vertical septum whose remaining portion forms the crucial ligaments.

At the beginning of the fifth month of intra-uterine life the hitherto complete intermediary disc softens in its central portion, and by the end of this month a gap has appeared on each side of the crucial ligaments. The absorption proceeds steadily, and at birth a definite semilunar gap is usually present in the central portion of each cartilage. The hiatus is bilateral, but is not of equal size on the two sides, that on the inner cartilage being much larger than the corresponding space on the outer mass.

ANATOMY AT BIRTH

At birth the semilunar cartilages cover most of the adjacent surface of the tibia and femur, the area of direct contact of the articular covered bone-ends being greater on the mesial than on the lateral aspect. Gradually the central gap increases in size and the area of direct bony contact reaches the usual adult proportions. This normal process of absorption of the central portion is occasionally deficient in the external cartilage, which then retains its fœtal arrangement. When this congenital abnormality is present the meniscus is otherwise normal, being much thicker on its outer border, becoming thinner as its central margin is approached, but showing little alteration in the regularity of its surface, with only a slight indication of the wave which develops later as a result of weight-bearing.

MINUTE STRUCTURE OF THE CARTILAGE

On section, the cartilage of the newly-born child shows three clearly differentiated zones, in each of which the cell formation is peculiar to itself, gradually merging into the type of tissue of its neighbour. The outer, or vascular zone, is composed largely of curling, wave-like, and a few straight connective-tissue fibres, together with scattered strands of elastic tissue. The blood-supply in this area constitutes its outstanding feature, vessels reaching it from the capsule and especially from a peri-meniscal artery, which sends branches into the middle and inner zones. At this period the vascular supply of the cartilage is very free, the vessels spreading from the outer border to the inner sharp margin. These vessels are derived from three main sources: the peri-meniscal artery with its radiating branches, which pierce the outer zone of the cartilage and spread as a fan through most of the cartilaginous substance; the two other arterial trunks reach the cartilage at its anterior and posterior bony attachments, where they are continuous with those of the crucial ligaments.

With the instigation of movements of the joint, changes appear in the cartilage and in its blood-supply. The separate wavy connective-tissue bundles become fused into a more resistant mass by a process of devascularization, the blood-vessels disappearing from the inner zone, diminishing in number and size in the middle and

* Robert Jones Memorial Lecture of the Royal College of Surgeons of England, delivered on Dec. 4, 1940.

neighbouring sector of the outer zones, and with the commencement of walking this process is completed in the middle and inner zones.

The elementary substance of the inner segment of the cartilage consists of undulating, coarse, oxyphil, collaginous bundles, between which lie a few fibroblasts with spindle-shaped nuclei with here and there a true cartilage cell. The elastic fibres give the cartilage its characteristic wavy appearance.

There is still considerable difference of opinion as to the exact period at which true cartilage cells can be demonstrated. The most authoritative work on the minute structure of the cartilage was published by Ichido in 1932, and according to his findings true cartilage cells are never found at birth, and do not appear until 6 or 7 months later. These findings have not been confirmed by me in a series of sections taken from the cartilages of newborn infants, in each of which true cartilage cells can be clearly seen as small localized islands.

The change into cartilage occurs first in the middle section of the central zone as a cloudiness in the connective structure, at first in small localized pockets which steadily increase in size. With this alteration the waviness of the cartilage disappears, its elasticity and sponginess diminishing with the increase of its stability. Eventually the inner and middle zones become entirely cartilaginous in structure, but this alteration never involves the outer zone, which retains its connective-tissue structure throughout life, and forms the obvious line of demarcation between the cartilage proper and the capsule of the joint.

CHANGES WITH AGE

Owing to the large proportion of elastic fibres the cartilage of the young child is extensible, and can be bent ot stretched without rupture, but as its elastic fibres disappear the cartilage becomes more rigid, and therefore more liable to injury. All these changes are normally completed at adolescence, only the outer zone retaining its loose vascular connective-tissue structure. As a rule, no further change occurs until the third decade, when signs of degeneration can occasionally be demonstrated. These may be of a fatty or a mucoid type, the latter being at first localized to one or more minute areas; in fact, only one fibre may be at first involved, the change slowly progressing to affect more and more of the cartilage substance. Near these areas of degeneration cell proliferation can be observed in the neighbouring healthy cartilage tissue. Occasionally in the midst of an area of mucilaginous degeneration phosphate of lime is deposited, at first hard to define, but later becoming more definite and extending through the whole area of degeneration. Fatty degeneration can also be demonstrated, but this change does not, as a rule, appear in the cartilages of people under 50 years of age, although the change may on occasion appear at an earlier date. In this process not only the cartilage cells, but the elementary basic substance is also involved. By this alteration the strength of the cartilage is greatly reduced, its normal glistening white appearance being converted into the dull creamy yellow cartilage of old age.

CARTILAGE LESIONS AND ABNORMALITIES

Changes Following Injury.—As a result of excessive strain a cartilage usually tears in its middle or inner zones, although occasionally the whole structure appears to be torn bodily from its attachment to the capsule. Following such an injury changes occur in the cartilage, the particular type of change depending to a large extent on the situation of the lesion. When the tear involves only the outer zone, reattachment of the cartilage in its normal position occasionally occurs owing to the free blood-supply which is present in this area. The possibility of reattachment depends on treatment; if rest of the injured tissues is adequate newly formed blood-vessels grow from the capsule into the cartilage, resulting in a firm fibrous tissue attachment, but without rest this regenerative process is interrupted and the rupture remains.

When the tear is present in the middle or inner zones the secondary changes are very different. Blood-vessels being absent in this area, union is impossible, and no instance of healing of a ruptured cartilage, or even of the occurrence of inflammation round a central cartilaginous tear, has ever been demonstrated. On either side of such a tear the cartilage cells proliferate, the nuclei increasing in size and number, but never producing fusion across the rupture in the cartilage.

When the injury consists of a split in the cartilage substance cell proliferation alone can be demonstrated, but if a portion is broken off and becomes detached, degeneration occurs rapidly in the separated fragment. The detached portion shrinks, its centre being converted into hyalin material, while the outer covering retains its original structure of fibro-cartilage, being nourished apparently by the synovia.

Mechanism of Cartilage Lesions.—There are two main methods by which the mechanism of cartilage lesions can be studied :—

1. By a study of the ligamentous and bony attachments of the cartilages, and by an examination of the effect of movements of the joint either at operation or in the dissecting room.

2. By a close examination of the history of the accident, especially in regard to the position of the joint and the movements of the limb at the time of injury, followed later by inspection of the cartilage itself at operation.

Both cartilages are firmly attached to the upper surface of the tibia at their anterior and posterior ends, the outer margin of the inner cartilage being also adherent to the deep short

fibres of the internal lateral ligament. Through the remainder of their outer margins the cartilages are loosely attached to the capsule by the connective tissue of the original vascular zone of the fœtal meniscus. Owing to their attachments the cartilages appear to be accessory to the tibia, but movements of the joint demonstrate that they alter their position with the movements of the femur rather than with those of the tibia. This fact is evident on inspection of the knee-joint at operation, the cartilages following in a restricted range the movements of the femoral condyles. During such movements it is also evident that on flexion or extension of the joint both cartilages follow the movements of the femur without strain, and with no alteration in their shape. This freedom of movement of the cartilages is considerably diminished if flexion and extension are produced while the knee is retained in internal or external rotation. In this position the cartilage remains closely adherent to the head of the tibia, unaltered in position by flexion and extension of the joint, whilst at the same time tension in the affected cartilage is greatly increased. The reason for this increase of tension also becomes clear; by internal rotation of the femur the middle of the attached margin of the internal cartilage is pulled backwards and inwards, while its anterior attachment is pulled forwards and outwards. At the same time the femur slides inwards from its normal articulating area on the head of the tibia, whilst the cartilage is pulled by its anterior attachment towards the opposite femoral condyle, across the middle of the joint. If, during this movement of rotation, the cartilage remained in contact only with the outermost margin of the femoral and tibial articular surfaces, the pressure exerted on the cartilage would be unchanged however great the increase in tension.

As the cartilage slides further into the joint its inner portion is gripped between the femur and the tibia, and a continuation of the rotation of the joint causes a rupture of its substance at one of the points of greatest tension. These points are the attachments of the cartilage to the head of the tibia, the inner free margin, and the substance of the cartilage itself, external to the point of greatest pressure between the femur and tibia. Examination of injured cartilages at the time of operation confirms the mechanical principals of cartilage lesions, the majority occurring at those sites where tension is at the maximum.

The same movement of internal rotation of the femur on the tibia produces a similar but lesser strain on the posterior attachment of the external cartilage, which being more mobile is less liable to injury. Again, in external rotation of the femur the conditions are exactly reversed, the strain is then applied to the anterior portion of the external, and to the posterior section of the internal, cartilage, so that from the mechanical point of view injury of either cartilage may occur, either with internal or external rotation of the femur on the tibia, although the site of the lesion is more or less rigidly governed by the direction and degree of the rotating force. Clinical evidence bears out the suggestion that cartilage lesions do occur in this manner; experimentally and clinically it is found that a lesion of a semilunar cartilage does not occur whilst the knee is fully extended, except where the cartilage lesion is only an incident in a very extensive destruction of the joint. In full extension rotary movements of the joint are impossible, and excessive abduction or adduction of the limb can only cause fracture of an articular surface, or rupture of the opposing lateral ligament.

In the last 200 operations carried out by me for lesions of the semilunar cartilages, in which especial care was taken to determine the exact anatomical abnormality, 68 per cent consisted of longitudinal tears of the substance of the cartilage, 7 per cent showed splits on the inner free border of the meniscus, whilst the remaining 25 per cent was made up of detachments of the posterior-external border of the external cartilage, free fringes from the medial edge and nodular excrescences of the cartilages, together with congenital maldevelopments of the external cartilages.

The proportion of lesions of the internal, as compared with those of the external, cartilage is of interest; in this series the proportion was 3·7 to 1, a figure which differs very considerably from many given previously.

Clinical Signs and Symptoms.—As the injuries sustained by the semilunar cartilages vary so greatly in severity and in position, it is obvious that the clinical signs must also vary. The classical story of an internal derangement of the knee consists in a history of strain of the joint followed by locking, which is, as a rule, repeated at intervals. Accompanying this locking there is pain over the site of injury, followed by tenderness, which may remain for several weeks, even after the displacement has been reduced. Such a history, associated with a normal X-ray appearance of the joint, is usually considered to be essential in making the diagnosis of a cartilage injury.

Locking of the joint can only be produced by the interposition of tissue between the articular surfaces of the tibia and femur in front of the mid-plane of the joint. Locking—the result of a cartilage lesion—therefore, can only occur in those rare cartilage injuries where the tear has occurred in the anterior section of the cartilage, or in the other much larger group in which a split in the cartilage substance extends through its length, the so-called bucket-handle tear. In the other cartilage lesions, in which the injury lies behind the midline of the joint, displacement of the broken fragment might produce a block to flexion, but could not prevent full extension. This blocking of flexion is seldom appreciated because kicking the leg into full extension is the invariable response to any abnormal sensation in the knee, and if this movement can be completed

the joint feels better, and any cartilage displacement in the posterior compartment is at once corrected. The sign of locking of the joint, on which so much stress has been laid, is, therefore, not present in all cartilage lesions, and this sign—although important—should be recognized as only one of a group useful in diagnosis. Again, a sensation of locking is not always due to cartilage displacements, but may follow any strain of the anterior part of the joint, the pain thereby produced causing a reflex inhibition of voluntary extension, thus simulating a true mechanical locking. Similarly, the loss of power of extension may be caused by the presence of a loose body, enlargement of the post-patellar pad of fat, fracture of the spine of the tibia, or osteochondritis dissecans, each of which must be considered before arriving at the diagnosis.

The presence of tenderness over a cartilage is an important clinical point, and suggests the possibility of a lesion of the underlying meniscus. The tenderness may be present along the whole course of the injured cartilage, but is, as a rule, most acute at one of three points. These are the regions of the anterior and posterior attachments of the cartilage, and, in the case of the inner cartilage, over the middle of the internal lateral ligament, whose deep fibres are at this point attached to its outer rim. Tenderness over one or other of the terminal attachments of the cartilage to the tibia suggests the presence of a strain or tear at this point, but the severity of the lesion cannot be gauged by the degree of tenderness; in fact, a strain may give rise to extreme localized tenderness, while a complete rupture of the cartilage at the same point may be comparatively free. Similarly, tenderness on pressure over the middle of the internal lateral ligament at the level of the joint may be taken as an indication of strain of the deep fibres of the ligament, but this sign of itself cannot be taken as a measure of the severity of the injury. The sign of localized tenderness should, therefore, be given weight in arriving at the diagnosis so long as it is remembered that a correct diagnosis can only be arrived at by balancing all the evidence. The absence of tenderness must not be taken as a contra-indication to the diagnosis of a cartilage lesion because, although usually present after the injury, it tends to diminish, and in many cases disappear, after a few weeks, even though the underlying cartilage may be broken or displaced.

In weighing the significance of localized tenderness greater reliance can be placed on this sign when it is present in the middle of the internal lateral ligament than when present over its anterior or posterior attachment. The explanation of this point can be found in the difficulty with which localized tenderness is differentiated in the region of the anterior and posterior attachments of the cartilages, where so many other tissues rather than the cartilage may have been injured. Turner, of Leningrad, has described an area of hyperæsthesia, which he states is always present over the antero-internal aspect of the joint in lesions of the internal semilunar cartilage, but I have never been able to confirm this sign. Although many of the signs and symptoms of cartilage lesions are present equally with injuries of either meniscus, many others are found peculiar to one cartilage, and it is advisable that the injuries of each cartilage should be considered separately.

THE INTERNAL CARTILAGE

As the inner side of the knee-joint normally forms the apex of the angle between the femur and the tibia, it follows that the strain thrown on this area is considerably greater than that on the outer aspect of the joint. Again, the internal cartilage, being attached to the internal lateral ligament, is more stable in position on the upper surface of the tibia than the outer, and has, thereby, thrown on it much greater strain, increasing the liklihood of serious injury.

The injury most commonly encountered in the internal semilunar cartilage is the longitudinal or bucket-handle split, usually found about mid-way between the free and attached margins, the inner segment, although equal in breadth to the outer, generally forming less than half its substance. Occasionally the inner segment is so large that it appears to include the whole of the cartilage, but a thin rim always remains attached to the capsule where it can be demonstrated at operation. In this type of injury the inner segment remains attached at its anterior and posterior ends, whilst its substance, being mobile, can pass between the articular surface of the femur and tibia into the intercondyloid notch of the femur, and if not reduced by voluntary efforts or by manipulation may remain displaced for many years, giving rise to little discomfort or disability beyond a very slight limitation of full flexion and full extension. The margins of the split become rounded and smooth, especially after frequent displacements, which tend to produce a diminishing degree of discomfort and synovitis on each occasion.

Flap injuries, in which the split in the cartilage extends into its substance from the free border, form the second largest group of cartilage lesions. They are found at any point from the anterior to the posterior attachment, with a predilection for the posterior segment of the joint. Thus, in 35 such lesions occurring in this series, 24 were situated posterior to the plane of the lateral ligaments, while only in 1 case could such a lesion be demonstrated close to the anterior tibial attachment. The split in the cartilage may extend in either direction, forwards or backwards, and the severity of the resulting disability depends naturally on the thickness of the pedunculated mass. In some instances, where the segment is very small, the symptoms are slight and indefinite, and the diagnosis is a matter of considerable difficulty. With such small flaps, even when situated anteriorly, locking of the joint is mechanically impossible, and nipping of the partially

separated mass produces only an indefinite feeling of instability in the joint. When the flap is larger, locking of the joint can occur if the segment becomes wedged in the anterior compartment of the joint. Occasionally, after a long history of disability caused by such a lesion, the fragment may break loose from its attachment and all trouble cease for a time. Such a loose fragment may later cause lockings at different points, or it may become attached to the capsule at some site where mechanical interference with the joint movements is impossible. As a rule, with extension of the split in the cartilage and consequent enlargement of the pedunculated mass, the resultant signs and symptoms increase in severity until a stage is reached at which the diagnosis is obvious.

THE EXTERNAL CARTILAGE

All the different forms of injury occurring in the internal cartilage may occur also in the external, but the proportions in which the types are found in the external cartilage do not correspond.

Bucket-handle splits of the external cartilage constitute only 30 per cent of the total, while pedicled lesions comprise between 20 per cent and 25 per cent of the remainder. The commonest pathological condition of the external cartilage consists in an almost complete detachment of its postero-external margin from the capsule, a condition which permits of abnormal mobility and nipping. The line of separation lies in or near the outer vascular zone, although cartilage tissue can usually be demonstrated as a thin ribbon attached to the capsule.

As the attachments of the external cartilage are not so complete as those of the internal, it escapes many of the injuries which occur to the inner cartilage. The signs produced by abnormalities of the external cartilage—whether they be congenital or acquired—show certain distinct characteristics, the first of these being the peculiar snap or 'clunk', which is accompanied by a momentary lateral displacement of the femur on the tibia. This characteristic 'clunk' is heard almost at the termination of full voluntary extension, while at the same time the whole joint appears to shiver, the lower end of the femur apparently sliding inwards on the tibia, and then back again to its normal position. The cause of this lateral slide of the bones can be appreciated during the removal of a loose external cartilage, the same jerky movement of the femur on the tibia being produced when the posterior portion of the cartilage is pulled forward between the articular surfaces. Neither the noise nor the movement is evident if the loose portion is small and pedunculated; apparently they can only follow the slipping of a large mass.

CYSTS OF THE SEMILUNAR CARTILAGE

The occurrence of cystic swelling originating from the semilunar cartilages has long been recognized. Although considered at one time as being derived only from the external cartilage, it is now recognized that it may be derived from either cartilage. These swellings are usually situated along the outer margin of the affected cartilage in close proximity to the adjacent lateral ligament, and for many years this was considered to be the only possible site. Recently, Ollerenshaw has described a cyst of the semilunar cartilage appearing on its inner free margin.

Two beliefs as to their origin are still widely held, the first that they are congenital in origin, the cavities being lined with what is described as endothelium, and the second that they are traumatic in origin, the lining wall being composed of flattened cartilage fibres, which are so thinned out by pressure that they bear a close resemblance to endothelium.

In many instances the history of previous direct trauma to the region is given by the patient, and the fact that the cysts appear in the adult when such trauma is probable, and not in the baby or young child, would seem to support the second theory.

The patient complains of a chronic, continuous aching in the region of the cyst; he states that, although constant, the aching is diminished by rest and increased by exercise. Synovitis is rarely present, nor are there any of the signs or symptoms usually associated with a cartilage lesion. In a few instances a concurrent tear of the cartilage is present, a circumstance which supports the traumatic theory.

ABNORMAL LAXITY OF THE SEMILUNAR CARTILAGES

Much discussion has arisen on the question whether a cartilage, which is normal in structure but to which the attachments are lax, can give rise to the signs and symptoms of an internal derangement of the joint. Such abnormal slackness of the cartilage is almost invariably a part of a general laxity; the muscular control of the joint is poor, the capsule has been stretched through frequently recurring synovitis, and all normal movements are exaggerated. Such a cartilage is able to glide abnormally on the tibia and, with rotation of the joint, there may be a momentary nipping of the meniscus sufficient to cause a sensation of locking. The condition can be differentiated easily from a true cartilage lesion; the locking, or nipping, is not complete, being simply a momentary stab of pain in the region of the cartilage, which may cause a temporary inhibition of the joint, but is never followed by the sensation of unlocking.

CONGENITAL ABNORMALITY OF THE SEMILUNAR CARTILAGE

As previously mentioned, persistence of the fœtal form of a semilunar cartilage occurs only in the external meniscus, and patients suffering from this abnormality come under observation

at an early age. Although the cartilage in these patients is complete, its inner unabsorbed portion is thin, and may show one small hole in its substance some distance from its inner margin.

The characteristic signs and symptoms produced by this abnormality are a feeling of discomfort, accompanied by a snapping or banging noise, as the knee is flexed and extended. As a rule the disability is insignificant, the patient, being accustomed to the noise since childhood, is not worried, and may not seek advice unless and until arthritis develops at a later date.

On inspection the congenitally abnormal cartilage shows a thickened ridge or wave on its upper surface just behind the depression in which the femur rests in the fully extended joint. Occasionally two such ridges can be demonstrated, one in front, and the other behind this point. In flexion the femur rolls backward over the ridge on the posterior part of the cartilage, while in the movement of extension the ridge is forced forward below the articular surfaces, causing the apparent shifting of the femur on the tibia. Removal of the entire cartilage is followed by cessation of the noise and of the abnormal movement.

METHODS OF EXAMINATION OF THE KNEE-JOINT

History.—In arriving at a correct diagnosis of knee-joint injury considerable weight must be given to the severity and direction of the injury sustained. If recurrence of the displacement has been experienced, was the pain and discomfort on each occasion felt at exactly the same point? Generally nothing unusual is felt round the joint, but 2 patients out of the 200 of this series stated that during the period of locking they felt an abnormal lump projecting under the skin in the region of the affected cartilage. This unusual projection of a portion of an injured cartilage can only occur when the partially detached portion has a long pedicle permitting the flap to be forced superficially.

A history of locking of the joint is not enough to clinch the diagnosis, the type of locking must be determined as well as the after-history, which is almost as important as the locking itself. By the word 'locking' the patient is simply describing an inability on his part to complete the full extension of the joint, usually from a point about 25° to 30° from full extension. The inability to extend the joint may be caused by many conditions other than the displacement of a part, or the whole, of a semilunar cartilage. Thus, strain of the capsule on the anterior section of the joint, or injury to the post-patellar pad of fat may give rise to pain which inhibits full extension of the joint, simulating very closely a true locking due to a cartilage lesion.

The outstanding differential point of these different clinical conditions is found in the subsequent history. In the first, where the locking is due to the presence of a section of cartilage between the bones, the locking is sudden and the unlocking is equally definite. When the displaced portion of cartilage has been restored to its normal position free movement of the joint occurs at once, and beyond a temporary tenderness the joint feels almost normal. The restoration of full movements after a strain or bruising of the soft tissues in front of the joint is a slow process, extending over days or weeks, and full movement may or may not be restored.

Palpation.—By palpation alone many abnormalities may be diagnosed. The palpation must be continued during the full range of movements of the joint, as only by this means can such conditions as slipping of one of the hamstring muscles round the femoral condyles or biceps tendon over the head of the fibula be fully appreciated. Although it might be possible to recognize either of these abnormalities by inspection alone, the diagnosis is simplified by palpation. Similarly, the slipping of any of the tendons round exostoses can be distinguished, even when these projections are minute.

Loose bodies, even when they do not show in the X-ray photograph, can occasionally be felt, particularly when present in the suprapatellar pouch, and the possibility of moving them from side to side makes the diagnosis clear.

Cysts of the semilunar cartilages also can best be diagnosed by palpation; they form rounded tense swellings diminishing in size on flexion of the joint, and increasing with extension of the limb. They are slightly tender to pressure, and occasionally feel as of almost bony hardness, appearing rather below the level of the cartilage from which they arise. They can be distinguished by their site and consistency from other similar swellings in the neighbourhood, such as bursæ which are occasionally present between the tendon of the biceps and the upper end of the fibula, and the rare cystic degenerative swellings derived from perisynovial tissues. These latter are not so well defined as the cartilage cyst, they are less tense in structure, and usually appear at a higher level.

The presence and situation of areas of tenderness can also be defined, their extent and degree appreciated, and an approximate estimate made as to the tissue involved. Tenderness may indicate the presence of an underlying cartilage rupture, but it may also indicate a simple ligamentous strain or tear which has produced a localized injury.

X-ray Appearances.—Much valuable information—in most instances of a negative type—can be obtained by X-ray photography; the photographs should be taken in three positions, antero-posterior, lateral, and oblique.

In many instances, such as osteochondritis dissecans, it is possible to reach a definite diagnosis only with the help of the X-ray photograph, and then only if the loose, or partially loose, portion of the femoral condyle contains bony

tissue. The same proviso holds true in the diagnosis of loose bodies, and when these are composed of cartilage alone the radiograph is of little help, but with even a trace of bony tissue their diagnosis is possible.

Arthrography and the Injection of Gases into the Joint.—These have proved to be of little value in diagnosis and their use has gradually been discontinued.

Manipulation.—From the history, and by a careful clinical examination, it is possible to diagnose most of the semilunar cartilage lesions in which the injury has occurred anterior to the lateral ligaments. Tears or displacements posterior to this point produce so few of the classical signs and symptoms that other methods of examination are necessary for their elucidation. In this connexion the use of manipulation of the injured joint has proved itself of value.

If the cartilage is injured in its posterior section locking of the joint does not occur, the only indication of the interposition of the broken segment of the cartilage being a momentary loss of control, usually accompanied by comparatively little pain. The patient can never localize the site of the occurrence, the usual statement being that there seems to be a sudden loss of power. Subsequently there is a mild temporary synovitis, and when recurrences are numerous even this slight effusion is absent. Such an indefinite history gives no clue as to the pathological condition present, and when this story is associated with muscular wasting the diagnosis of muscular insufficiency is usually arrived at, and exercises and massage are prescribed.

If the cartilage is torn longitudinally the signs are at first indefinite, but become more obvious if the tear extends into the anterior segment of the cartilage. The method of examination by manipulation is of little value when the lesion is anterior to the midline of the joint, the area in which clinical signs and symptoms are, as a rule, so well defined that accessory methods of examination are not of paramount importance.

In carrying out the manipulation with the patient lying flat, the knee is first fully flexed until the heel approaches the buttock; the foot is then held by grasping the heel and using the forearm as a lever. The knee being now steadied by the surgeon's other hand, the leg is rotated on the thigh with the knee still in full flexion. During this movement the posterior section of the cartilage is rotated with the head of the tibia, and if the whole cartilage, or any fragment of the posterior section, is loose, this movement produces an appreciable snap in the joint. By external rotation of the leg the internal cartilage is tested, and by internal rotation any abnormality of the posterior part of the external cartilage can be appreciated. By altering the position of flexion of the joint the whole of the posterior segment of the cartilages can be examined from the middle to their posterior attachments. Thus, if the leg is rotated with the knee at right angles the cartilages in their mid-section come under pressure, but, anterior to this point, the pressure exerted on the cartilage is so diminished that accurate examination is impossible. When a loose segment of the cartilage is caught between the bones during the rotation, the sliding of the femur over the loose fragment is accompanied by a thud or click, which can sometimes be heard but can always be felt, and the size of the detached portion can be judged by the rocking of the tibia, and usually also by the severity of the sound produced.

This method of examination is not easy to master; the rotation requires a considerable amount of practice, and the whole procedure must be carried out systematically if success is to be attained. Probably the simplest routine is to bring the leg from its position of acute flexion to a right angle, whilst the foot is retained first in full internal, and then in full external rotation. Any abnormality in the cartilage structure in the area under examination will be discovered during the straightening of the joint.

In a child, and in some adults with abnormally lax cartilaginous attachments, clicks can be produced during this manœuvre, even in the absence of any cartilage lesion. There is little difficulty in differentiating these clicks due to excessive laxity from those caused by fragmented and loose portions of the cartilage. When the click occurs with a normal but lax cartilage, the patient experiences no pain or discomfort, but when produced by a broken cartilage, which has already given trouble, the patient is able to state that the sensation is the same as he experienced when the knee gave way previously. In examining patients with strongly developed thigh muscles difficulty may be experienced in rotating the leg, and in a few exceptionally strong or highly nervous patients, it may be advisable to give an anæsthetic in order to gain the necessary muscular relaxation.

The method, when correctly applied, gives very valuable evidence as to the existence of injury to the posterior segment of either cartilage. Not only is it possible to determine whether a lesion is present or not, but also to define accurately its site and extent, two points of diagnosis which are essential if the correct treatment is to be employed. The method is not described as a counter to the ordinary methods of examination of the joint, but rather as a useful accessory. Thus, if the patient has suffered from repeated lockings of the joint, indicating a lesion of the cartilage in its anterior section, and at the same time a definite click can be produced from the same cartilage posteriorly, it is evident that a bucket-handle tear is present. Again, if the tear is confined to the posterior section of the cartilage, and the diagnosis is made by the manipulation, the surgeon can with confidence remove the cartilage, even though on opening the joint there is no sign of injury in its anterior section.

TREATMENT

On this point there can be little discussion. After the first injury rest of the joint may be employed in the hope that the injury has occurred in the outer vascular zone, and that, as a result of rest, reattachment may occur. If recurrence has taken place further rest is inadvisable, and removal of the injured cartilage should be undertaken without delay.

Reasons for Failure of a Cartilage Operation.—When an injured semilunar cartilage has been removed by operation from a knee, which is otherwise normal, the result is usually so successful that it is profitable to consider the possible causes of failure. Leaving out of consideration such avoidable tragedies as septic infection, the first and most common error is undoubtedly incorrect diagnosis. Reliance on the site of pain or tenderness may lead the surgeon to remove the normal and leave the affected cartilage; in abdominal surgery the operation of laparotomy is often a justifiable procedure, but in a knee-joint, where internal derangement is suspected, arthrotomy must be condemned. If the lesions of a semilunar cartilage occurred always in the anterior segment of the joint there would be some justification for the procedure, but the frequency with which the tear is found in the posterior segment of the cartilage condemns the procedure.

When the knee-joint is opened on the anterior aspect, and the suspected cartilage appears normal, its removal can be undertaken with confidence only if the diagnosis of a posterior tear has been arrived at prior to the operation. A far too common error is shown in the incomplete removal of the injured meniscus; many surgeons are content when at operation they remove the broken portion of the affected cartilage, leaving in position the larger mass which is apparently firmly attached to the capsule. The partial operation is followed by apparent success in about 70 per cent, no further disability being complained of, and the patient being perfectly satisfied. This type of operation is not a sound surgical procedure; in those patients who continue an athletic life recurrence of symptoms is the rule rather than the exception. The 70 per cent previously mentioned, is composed largely of those who, after the operation, do not continue with the more strenuous athletic pursuits and take up some other less active pastime. A patient in whom part of the cartilage has been left, often complains of a feeling of weakness on sudden twists of the joint; locking does not occur, but the knee feels unstable and does not permit full activity.

If such a joint is examined by manipulation the femur can be felt sliding off the thick edge of the cartilage which has been left in position, the movement giving rise to a feeling similar to that experienced by the patient on using the joint. The prevention of such failures lies in the removal of the affected cartilage and not only of its detached or pedunculated fragment.

The length of the incision used for removal of the cartilage affects the after-result; in any form of surgery the incision should be so planned and of such extent that the operation field is readily accessible and can be inspected freely. This principle is excellent, but the greater the experience of the surgeon the more easily can he obtain his requirements through a comparatively small opening. Long incisions in the knee-joint are not without their disadvantages; as an illustration of this it has been necessary on two occasions to treat a patient for recurrent dislocation of the patella, developed from stretching of an abnormally long operation scar on the inner side of the joint. Occasionally following operation on the knee the patient suffers from persistent synovitis, which is in my experience much more frequent where the operation scar is extensive and where unnecessary trauma to the tissues of the joint has occurred. Clamps should never be applied to any of the tissues in the joint, as the response to such crushing appears invariably as a post-operative synovitis. The tissues should be retracted gently with a smooth, flat retractor which does not injure the superficial layer of the synovial membrane, and sutures should be used sparingly in the joint cavity; irritating tissue, such as chromic gut, is to be avoided.

Physiotherapy has an important part to play in the after-care of the knee, exercise of the quadriceps is essential, and massage is excellent so long as it is used intelligently. When applied to the knee itself massage only causes irritation, but massage of the thigh above the joint improves the circulation, improves the muscle power, and prevents or disperses post-operative synovitis.

THE TREATMENT OF CARDIOSPASM BY THE HELLER TYPE OF OPERATION

WITH SPECIAL REFERENCE TO THE CHOICE OF OPERATION, ITS INDICATIONS AND TECHNIQUE, AND WITH A REPORT UPON THREE PERSONAL CASES

By DONALD BARLOW, London

SOME GENERAL CONSIDERATIONS

Walton[1] quotes the following clear definition of cardiospasm: "A condition of dilatation and hypertrophy of the œsophagus where, on post-mortem examination, no obstruction can be found distal to the dilatation". This definition, although important from the pathological point of view, is open to the obvious objection that it does not assist the clinical diagnosis. From time to time statistics are confused by the inclusion of other diseases, notably carcinoma, hysterical spasm, and dilatation of the œsophagus secondary to organic stricturing of the lower end of that organ or even of the stomach or pylorus. Barton and Dent's[2] case of the latter is of particular interest.

It cannot be too strongly emphasized, therefore, that before any form of treatment for cardiospasm is instituted, a most careful and full investigation is essential to establish the diagnosis beyond doubt.

The weight of evidence seems to point to two main primary conclusions with regard to cardiospasm, namely:—

1. The disease is due to a disordered neuromuscular mechanism at or below the level of the diaphragm. The exact nature of this disorder is uncertain, but seems to be a condition of spasm rather than inco-ordination of the peristaltic wave.

2. There is reason to believe that the lesion is congenital in origin.

In addition, two important secondary considerations are the complications which tend to follow, in varying degrees of severity, as a direct result of the primary obstruction: (1) Œsophagitis; (2) Lengthening and sagging of the œsophagus.

Successful treatment must effectively and permanently overcome both this obstruction and its concomitant complications and the choice of operation must fall upon the safest way of doing this. There seems no doubt that there are several good methods of attack, all of which have apparently been followed by excellent results. The statistical results of the three most promising vie with one another for the premier place (*see below*) and the writer is handicapped by having tried only one method, and that on relatively few cases. None the less, there seem to be definite reasons for preferment of the operation advocated in this paper.

Before going into these, it seems advisable to elaborate the four cardinal factors and to see what conclusions may reasonably be drawn from them by way of corollaries:—

a. Treatment, failing a really satisfactory operation upon the nervous control, must encompass the total division (or rupture) of the muscle of the spasm area (namely of the region from just above the diaphragmatic level to the top of the stomach) at the same time running the minimum risk of rupturing the mucosa. Knight[3] has devised an operation upon the sympathetic nervous control, but it is open to the objection that sympathetic nerve-fibres tend to enter the region from above the diaphragm.

b. With reference to the congenital nature of the disease, radical treatment should usually be undertaken at an early date after the onset of symptoms. Cases divide themselves into two natural groups: those starting early in life—9 cases are recorded before the age of five; and those starting commonly after middle age. There is no doubt that in neither group is there any tendency to natural cure, and, if neglected, the disease progresses steadily to a fatal termination. Many instances are mentioned in the literature of quite advanced changes in the œsophagus when symptoms are still very slight, and indeed the disease has been noted accidentally during routine radiography, long before symptoms appeared at all.

c. The extent of secondary inflammatory change in the lining of the œsophagus is not necessarily proportional to the length of history. It is a very important factor in designing treatment because it causes the mucosa to become even more friable than is normally the case and is the principal potential source of infection. Mediastinitis accounts for nearly all the post-operative deaths.

Whatever operation is undertaken, it is generally agreed that pre-operative lavage is beneficial, although doubt is sometimes expressed whether it is always possible to get the flask quite clean.

d. Œsophageal lengthening and consequent sagging, usually over the right side of the diaphragm, are potent causes of failure or of only

partial success of operations designed solely to dilate the obstructed area. Von Hacker[4] indeed regarded this as the principal cause of failure in certain types of case and designed an operation especially to overcome it. None the less the dilatation operations from above or below ignore it completely, although Walton, Shattock,[5] Lambert,[6,7] Moore,[8] Brown Kelly,[9] and many years ago Mikulicz,[10,11] have all stressed its great importance. The fact is that, where no reservoir is present, several types of operation may succeed, but where there is definite sagging, this must be taken into account, even though it be present only in a minor degree.

INDICATIONS FOR TREATMENT IN GENERAL

In practice, all degrees of the condition exist, from symptomless dilatations passing unnoticed or almost unnoticed until discovered radiologically, through those which, in spite of difficulties or discomforts in swallowing, are compatible with a good general condition. At the other end of the scale are two groups which together make up what may be called the "established" cases. There are :—

a. Uncomplicated cases where bougies will generally go easily through the cardia, but which, none the less, may have severe symptoms.

b. Cases complicated by organic contracture or by a large dilatation and sagging of the flask and in which the most careful efforts at dilatation even under direct vision are liable to fail or remain ineffective.

In deciding treatment in an individual case, it is of the first importance to base the decision on *clinical grounds*.[12] It is true that radiology and œsophagoscopy may help in a subsidiary way, but the cardinal questions to be answered are :—

a. How much dysphagia is present and how much does it interfere with the patient's life?

b. What is the state of the nutrition in relation to the duration of the symptoms?

c. What is the mental make-up of the patient and is he or she likely to tolerate the regular passage of bougies or likely to be able to learn how to do this personally?

d. Will bougies pass easily and how much relief do they give?

Appreciation of the fact that the disease is probably congenital in origin and certainly shows no tendency to spontaneous cure leads one to stress the advisability of early and effective operation in the majority of cases. It must be conceded, however, that, since this is not entirely without risk and in view of the undoubted fact that certain patients are able to carry on a more or less normal life for many years with the aid of bougies, the above questions should be faithfully answered in each case.

For this reason, the technique of the bougie method of dilatation, as well as Plummer's[13,14,15,16] more forcible method, is included in this paper. None the less, it is the writer's opinion that a far greater percentage of cases than at present undertaken should be submitted to operation not only because the repeated passage of bougies and much physical and social misery are avoided, but because a chance of rapid and permanent cure is afforded by a relatively simple and safe operation.

TREATMENT BY DILATATION

Technique of Dilatation by Bougies.—There is no doubt that, provided a careful technique is adopted such as that advised by Guisez,[17,18,19] a number of early or tolerant uncomplicated cases do well on routine dilatations, and although a complete cure may be lacking, such patients are at least enabled to derive comfort compatible with normal life.

In contrast to the two other methods of dilatation discussed below, the bougie treatment does not aim at rupture of the œsophageal musculature. As with any other narrowed channel, safe dilatation must be both gentle and slow. Not more than two or three bougies should be used at one sitting and a beginning should always be made with a size smaller that that reached previously. At the initial catheterization, it is as well to use direct vision through the œsophagoscope and if difficulty is encountered, to cocainize the cardiac region with a swab on a holder. If there is still difficulty, Guisez's *cathéterisme en fasciceau*, using three or more filiform bougies in a manner similar to that used in urethral catheterization, may enable a start to be made, as one of the bougies will probably slip through. This initial filiform bougie may be left in for twenty-four hours.

In a similar way, when dilatations have been well established and it is felt desirable to enlarge the cardia to its fullest size, multiple small bougies are much preferable to the use of a single large one because the former method avoids the discomfort occasioned by compression of the larynx, and in any event a number of small bougies is better as the œsophagus dilates most readily in the transverse direction. Mercury sounds have benefited a large number of cases, but because of their weight tend to give discomfort unless held back carefully. In addition they are apt to increase sagging of the pouch, so that a time comes when no bougie of any kind will pass except under direct vision.

It is pertinent to mention here that, where it is decided to use the method of dilatation, routine œsophageal lavage is essential in order to avoid œsophagitis which might lead secondarily to stricture, increase of the spasm, or possibly new growth. With reference to diet, no special advice is usually necessary since patients know themselves what they can and cannot take. Meals should be frequent and small and the food well cut up.

Technique of Plummer's Hydrostatic Dilatation.—Plummer has perfected a simple

and ingenious method of forcible dilatation of the lower end of the œsophagus which has been extensively adopted at the Mayo Clinic, and this method is claimed to afford cure or relief in a large proportion of cases where symptoms are severe or the passage of bougies difficult.

The technique consists in the swallowing of a long length of twisted silk the end of which may be assisted through the cardia by the attachment of lead shot. Twenty-four hours after the silk is properly anchored, a No. 60 French sound is threaded on to it and thus guided through the cardia. The preliminary use of

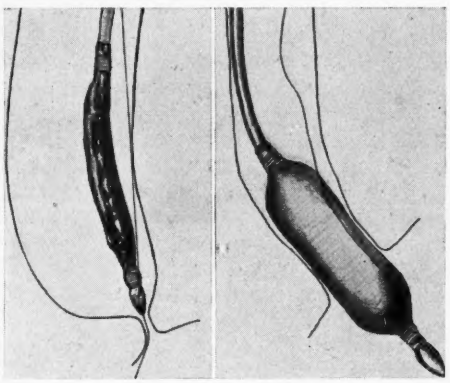

FIG. 359.—Diagram illustrating the passage of Plummer's hydrostatic dilator. (*After Moersch.*)

this bougie has the double advantage of making the dilatation more gradual and at the same time affording an estimate of the distance of the area of cardiospasm from the teeth so that the hydrostatic bag (*Fig.* 359) can subsequently be placed accurately.

Two or three days later, the bag is introduced in the same way as the bougie and is connected by a small pipe to a water tap. Water pressure is gradually exerted upon the cardia and this is controlled by a manometer. The degree of pressure necessary is said to vary according to the degree of dilatation of the œsophagus above the spasm, but in practice it is usually controlled by the sensation of pain. This pain, however, does not always occur. Pressures of from 20 to 30 ft. of water are generally held to be safe.

Gray and Skinner[20] report that of more than 1200 cases of cardiospasm encountered at the Mayo Clinic, mechanical dilatation by bougie or hydrostatic bag was carried out in the vast majority. No clear mention is made either in their paper or in that of Moersch[21] of the relative number of cases treated by Plummer's method and those merely receiving the passage of bougies. Of those treated by the hydrostatic bag, 9 are said to have died through the œsophagus having been split, but it is said that all these occurred before 1926 when the use of the preliminary bougie was first introduced.

In a series of 810 cases treated by one or other method of dilatation, and reported by Moersch, he mentions cure in 71 per cent and adds that repeated dilatations are usually not necessary. In the writer's experience with bougies, repeated periodical dilatations have always been necessary, but Moersch's statement may refer only to the hydrostatic method.

However this may be, the following criticisms of Plummer's method seem justified: (1) Cure is uncertain. (2) The operation must of necessity be a 'blind' procedure, and is therefore not in accordance with the best surgical principles. (3) There seems no method except the patient's sensation of pain of judging accurately what hydrostatic pressure is required in an individual case. Much experience is necessary to avoid splitting the œsophagus, and the method would probably be dangerous in inexperienced hands. (4) It does not cure those cases in which sagging has occurred.

TREATMENT BY OPERATION

Apart from the teachings of Walton, Shattock, and a few others, the full possibilities of surgical treatment for cardiospasm seem never to have been fully appreciated in this country. It is thought that the reason for this is the supposition that surgical access to the cardia is a procedure requiring unusual skill on the part of the operator and extreme risk for the patient. It is true that such operations as œsophago-gastrostomy either by the transpleural or the abdominal route, œsophago-cardioplasties, and the like are all fraught with considerable risk because of the technical difficulties of performing anastomoses near the diaphragm and because of the danger of sepsis.

Of the remaining procedures, two appear to bear analysis: Mikulicz's digital dilatation through the opened stomach; and Heller's[22,23] operation modified as suggested below.

Mikulicz's Digital Dilatation.—This operation, advocated by Walton in this country, is performed through a right pararectal incision, the œsophagus having been previously cleaned and emptied as far as possible and a tube left in situ. After carefully packing off the peritoneal cavity, the hand is introduced into the inside of the stomach via a longitudinal incision through the anterior surface of the organ. The cardiospasm is then gently dilated from below by the passage first of the index and later of the middle, ring, and finally of the little fingers. Special stress is laid by Walton upon the advisability of the use of the actual fingers rather than an instrument for the dilatation, because the sense of touch is an added precaution against rupture of the viscus.

In all, he reported 14 advanced cases treated in this way. Of these one died of heart failure on the seventh day after operation and one was relieved but still had to have bougies passed occasionally. All the remainder were free from symptoms for follow-up periods varying from 4 months to 20 years. These highly successful results cannot be criticized.

As with hydrostatic dilatation, however, there are several disadvantages with the Mikulicz operation: (1) The stomach has to be widely opened (the invagination method is not satisfactory) and there is therefore some risk of sepsis. (2) The cure depends upon a forcible stretching of the spasm, probably rupturing the muscle. Although this can be carefully controlled by skilled hands, no actual view is obtained of what is being done. (3) No provision is made for those cases in which sagging has occurred.

THE MODIFIED HELLER OPERATION

The principal, illustrated in *Fig.* 360, upon which Heller's extramucous cardio-œsophagotomy and the foregoing procedures are based, is that, provided sphincter muscle is intact, its contraction will narrow the contained lumen. If, however, it is split longitudinally, its contraction will not narrow but will rather widen the lumen, forming a gap through which the mucosa will bulge. With Heller's method the muscle division is done under direct vision and therefore tends to be both safer and more thorough than by blind stretching. The difficulty of combating œsophageal lengthening and sagging is met by incorporating where necessary von Hacker's idea of mobilizing the lower end and drawing it downwards into the abdomen to shorten and straighten the thoracic portion. Indeed this simplifies the procedure in all cases and is part of the suggested routine.

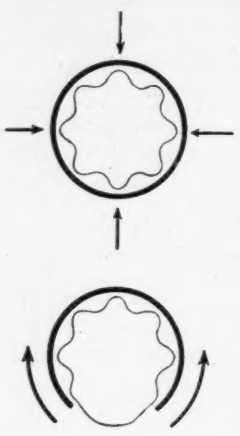

FIG. 360.—Illustrating diagrammatically the effect of division of circular muscles.

Walton's contention that Heller's operation was amongst those which were "unquestionably very severe" was hardly justified even in the original form in which the operation stood, for in 1914 Heller, in reporting his original successful operation, mentioned 16 other cases operated upon by various surgeons without fatality and with very good results in 12. In Holland, Zaaijer[24] states that Heller's operation was the normal method used, and mentions 8 successful cases without mortality.

The chief difficulty and danger which is liable to be met is the extreme friability of the lower œsophageal mucosa, often associated with adherence of the latter to the circular muscle. This is due to œsophagitis and can to some extent be lessened by pre-operative lavage.

The following modifications of the original operation are suggested, and, done in this simplified form, the exposure is excellent and the actual technique, beyond the vital necessity of care with the actual muscle division, is very simple and takes not much longer than the repair of an ordinary inguinal hernia:—

a. There is no necessity to turn up the costal margin.

b. No posterior longitudinal split in the œsophageal mucosa is necessary.

c. No opening is made in the stomach and no grooved director for cutting down upon is needed.

d. In *all* cases the œsophagus, which is easily freed from the diaphragmatic opening, is brought down at least two inches, thus improving the exposure and combating sagging.

Technique.—

Pre-operative Treatment.—Prior to operation, the contents of the œsophagus are drawn off, measured, and examined. An œsophagoscope is then passed and the mucosa examined. Lavage is then carried out twice daily, using sodium bicarbonate (1 drachm to 1 pint of sterile water) and later saline, as long as acute inflammation is present.

Operation.—The patient, who is usually thin because of loss of weight, is placed flat in the dorsal position with the bridge a little raised. The abdomen is opened through a left upper paramedian incision about 4 in. long.

The assistant's left hand is then put flat on the surface of the stomach with the tips of the fingers near the cardia, and gentle pressure exerted so as to compress the organ and to draw it downwards as far as possible. The left lobe of the liver is held upwards and to the right by a long-bladed (Diever's) retractor. In this way, a very good view of the cardiac area is obtained and the left gastric artery is seen. A transverse incision 3 in. long is made through the peritoneum (left triangular ligament) and the left vagus nerve identified and retracted medially (*Fig.* 361). Several small veins (branches of the left inferior phrenic and left suprarenal veins) surrounding the diaphragmatic opening are ligated and divided. At this stage the latter can be clearly identified and it will be found that with the aid of a blunt dissector, the œsophagus can be easily freed from it and lowered progressively into the abdomen. If there is œsophageal lengthening, von Hacker recommended suture in this position to the diaphragm.

By blunt dissection of the longitudinal muscle anteriorly and *extremely careful* cutting through of the thin circular fibres, the œsophageal mucosa appears at the bottom and bulges outwards. On its surface are one or two little submucous vessels which require ligation and division. *The incision should extend at least 2 in. on the œsophagus and 1½ in. on the stomach* (*Fig.* 362). The danger of the probable adherence and marked friability of the mucosa has already been emphasized.

The abdomen is closed without drainage unless the mucosa has been inadvertently damaged. In that case, the rent is easily seen and can be

sutured, a drainage tube being placed down to the site of operation.

Post-operative Treatment.—Sterile glucose water is given for the first twenty-four hours. Small and frequent fluid and semi-fluid feeds are increased over the next 8 to 10 days, when an increasing diet is instituted. Full meals are allowed after three weeks. Post-operative lavage does not appear to be necessary.

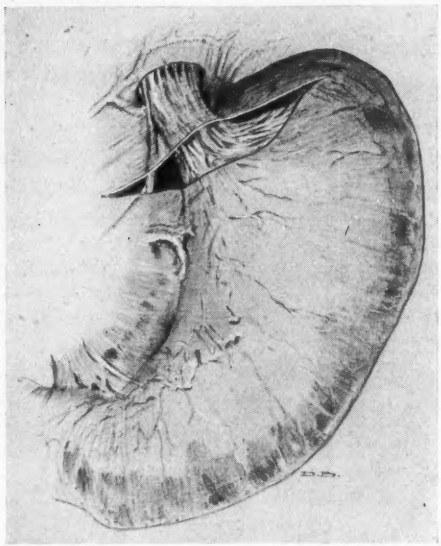

FIG. 361.—Modified Heller's operation. First stage.

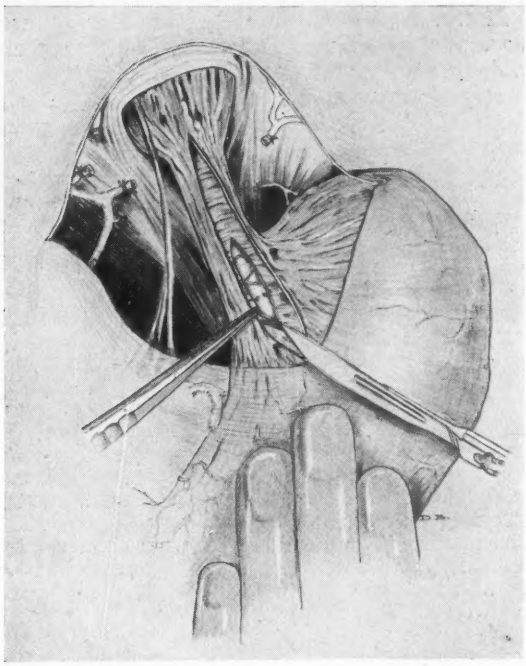

FIG. 362.—Modified Heller's operation. Final stage.

Late Results.—There is no doubt at all that in the vast majority of patients operated upon in this way there is a rapid and lasting functional improvement. Except in a very few of the early operations the writer has been unable to find records of recurrence of the symptoms. In some recorded instances and in one of the personal cases, there was a slight tendency to regurgitation when lying flat in bed because of the persistently open cardia, but this was easily stopped by giving the patient an extra pillow.

There is one rather remarkable point which has been repeatedly noted and which is demonstrated

FIG. 363.—*Case* 1. Radiograph taken before operation, showing dilatation of the œsophageal flask and narrowing of the cardia.

FIG. 364.—Same case. Radiograph taken six weeks after operation. Dilatation less marked and "might be regarded as normal".

to some extent in the accompanying radiographs (*Figs.* 363, 364). Although swallowing remains normal, the radiological appearance may seem disappointing. Delbet said in this connexion: "The satisfaction of the radiographer is often less than that of the patient".

However this may be, the simplicity of the extramucous cardiocœsophagotomy and the truly striking relief it brings to the sufferers whose comfort and social life have been for years a source of misery, seems to warrant its more general adoption.

CASE REPORTS

Case 1.—E. M. A., a married woman aged 39, with four children, was admitted to St. John's Hospital, Lewisham, on July 12, 1940.

HISTORY.—Four and a half years ago, the patient began to have severe "dragging" epigastric pain immediately after meals. The pain went through to her back and there was a feeling "as if a good deal of the food did not go through to the stomach". Very shortly after food, profuse vomiting occurred and the vomitus contained undigested portions of previous meals. Since the onset symptoms became progressively worse, although there were occasional periods of relief lasting a few days. During the last two years she had not even been able to swallow water and her life and that of her family had become miserable. She stated that the feeling of obstruction was worst at times of nervous tension. The bowels were opened fairly regularly and there was no cough or dyspnœa.

ON EXAMINATION.—General nourishment fair. No other abnormality found. *Œsophagoscopy* was omitted in error, but repeated lavage was given prior to operation.

Radiological Examination (opaque swallow).—Typical cardiospasm, with dilatation of the œsophageal flask and narrowing of the cardia (*Fig.* 363). Prolonged delay before any of the fluid seen to pass.

OPERATION.—Under nitrous-oxide—oxygen—ether anæsthesia, the modified Heller operation was performed on July 17. Wound closed without drainage.

PROGRESS.—The patient was discharged from hospital two weeks after operation. All dysphagia disappeared immediately and there has been no recurrence up to the present time. She reports that she "can now eat and drink anything without trouble" and she has put on weight. A radiograph (*Fig.* 364) taken six weeks after operation showed that "barium passed into the stomach without delay. The dilatation of the lumen was less marked and regarded as normal".

Case 2.—L. P., married woman, aged 63, was admitted to the Southend General Hospital on May 4, 1939.

HISTORY.—For the past nine years the patient had suffered from a "heavy" pain in the chest behind the sternum, much difficulty in swallowing, and regurgitation of food. During the last two years she had some relief by the regular passage of bougies and actually put on some weight. Lately, however, the pain and dysphagia had been worse and her general condition had deteriorated. She stated that she had private worries which usually made her worse.

ON EXAMINATION.—The patient appeared depressed and thin. Epigastric tenderness was present and there was a scar of an old uterine operation on the lower abdomen.

Œsophagoscopy.—This was carried out several times. There was considerable œsophagitis which cleared up fairly well with lavage.

Radiological Examination (opaque swallow and meal).—Moderate degree of dilatation of œsophagus due to cardiospasm. Partial obstruction at the lower end, but no evidence of malignancy.

OPERATION.—Modified Heller operation carried out on May 23, and wound closed without drainage.

PROGRESS.—Patient made an uninterrupted recovery and was discharged on the seventeenth day. All symptoms disappeared immediately and any type of food will pass without difficulty. There is a slight tendency to regurgitation if she sleeps with only one pillow. Radiographs taken five months after operation showed "no marked delay".

Case 3.—A. M. F., married woman, aged 65, was admitted to the Luton and Dunstable Hospital on Sept. 9, 1940.

HISTORY.—Difficulty in swallowing over a period of seventeen years—sometimes improving a little and then becoming as bad as ever. She stated that she "felt a spasm" when she had food and that she "knew when it opened". The original onset had been sudden. Sometimes she would go all day and even two or three days and then abruptly it would open. She dare not have a proper meal and had had no meat for a long time. She mostly had to be content with bread and butter, milk puddings, and an occasional soft-boiled egg. At times the "sweat would roll off her" in trying to get something through. Vomiting of food and slime occurred an hour or two after eating unless the spasm gave way. She had no special worries apart from her inability to meet people socially. She had used mercury bougies for many years, but without more than very transient relief.

ON EXAMINATION.—Thin woman with no abnormal physical signs. Scar of old cholecystectomy operation.

Œsophagoscopy was not done. Repeated lavage was necessary before all mucus and food was got away.

Radiological Examination (opaque swallow).—Typical cardiospasm with narrowing of the cardia and dilatation of the œsophagus above. A little barium passed through.

OPERATION.—The modified Heller operation was carried out on Sept. 16. In this case very considerable difficulty was experienced partly because there was a gross perisplenitis with multiple adhesions around the cardia, and partly because the œsophageal mucosa was very adherent to the circular muscle so that it was almost impossible to separate the one cleanly from the other. This was finally achieved, but two small holes were made through the mucosa. These were closed with fine catgut sutures. The abdomen was closed with drainage.

PROGRESS.—Healing of the upper end of the wound was delayed for several weeks because of a little leakage from the œsophagus. This finally healed soundly. From the time of operation, all discomfort and dysphagia disappeared. She has put on weight and has resumed her many social activities.

Since the completion of this paper, Mr. Kenneth Heritage (London), has told the writer of a case of his own which, following an earlier conversation, he had successfully operated upon in the manner suggested. A previous Mikulicz operation had failed to bring relief. Mr. Heritage has kindly consented to the publication of his notes:—

Case 4.—W. C., aged 31. In the summer of 1936, at the age of 27, the patient first noticed gradual onset of dysphagia, as if there was a "lump in the chest". The condition got gradually worse, until September, 1937, when he applied to the Princess Beatrice Hospital.

September, 1937.—Barium swallow showed a moderate degree of cardiospasm. The cardiac orifice was dilated by bougies and by the passage of the œsophagoscope under local anæsthesia without improvement, so at the end of October, 1937, Mikulicz's operation (transgastric digital dilatation of the cardiac orifice) was performed. No organic lesion was found at the cardiac orifice and four fingers were introduced for dilatation.

In spite of this, dysphagia was not improved, and from leaving hospital the obstruction became more pronounced and accompanied by a heavy sensation in the chest, necessitating self-induced regurgitation of œsophageal contents. "He could not eat any meal without having a glass of water handy to sip with his food to help it down".

August, 1940.—Barium examination of the œsophagus now showed slightly more marked degree of obstruction.

September, 1940.—Abdominal exposure of subdiaphragmatic portion of the œsophagus. Peritoneum over it divided. Œsophagus mobilized after ligature of coronary branches. Œsophagus delivered from diaphragmatic orifice and upper dilated portion brought down so that an incision could be made on its anterior surface extending from 3 in. above to 2 in. below the cardiac orifice. Muscular coat was thus split, exposing the mucosa in the whole length of the incision. Abdomen was then closed.

Since this date the patient has had very little trouble with swallowing, has lost the feeling of weight in the chest, and never regurgitates. He can swallow solids and liquids without difficulty. Barium examination shows only slight residual dilatation apparent only when thick paste is used and not demonstrable with fluid meal.

March, 1941.—Œsophagoscopy under local anæsthesia shows no obstruction whatsoever. The site of the cardiac sphincter cannot be identified.

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EXPERIMENTAL SURGERY

OBSERVATIONS ON THE CHOLEDOCHO-DUODENAL MECHANISM AND THEIR BEARING ON THE PHYSIOLOGY AND PATHOLOGY OF THE BILIARY TRACT*

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During the last twenty years considerable attention has been directed to the activities of the normal and pathological gall-bladder; to the mechanism controlling the discharge of bile into the intestines; and to states of motor dysfunction in the bile-passages—a condition known as biliary dyskinesia. It is recognized that such motor dysfunction may be responsible for typical symptoms of gall-bladder disorder, yet the patient will present at operation an apparently normal biliary tract. Alternatively, the symptoms may appear as a delayed post-operative complication of a cholecystectomy.

That biliary dyskinesia is a result of spasm about the lower end of the common bile-duct, with a resulting rise of pressure and distension in the biliary passages, is now generally accepted as a result of recent clinical research work. The exact site of the spasm, the cause of its onset, and its relationship to pathological processes in the biliary system have not been satisfactorily explained. The desire to obtain further information on these problems prompted the undertaking of this experimental investigation.

THE LITERATURE

It is apparent that an understanding of the normal working of the choledocho-duodenal mechanism and its controlling factors are an essential part of any experimental work which aims at elucidating the aetiology of biliary dyskinesia. For this reason a brief survey of the literature relating to the applied physiology and pathology of the biliary tract was made.

For purposes of clarity the literature is reviewed in four groups: (1) The preliminary observations of the late nineteenth century; (2) Anatomical contributions; (3) Physiological contributions; (4) Contributions on biliary dyskinesia.

Preliminary Observations of the Nineteenth Century.—The first descriptions of a sphincter around the lower end of the common bile-duct came from Glisson in the seventeenth century and from Gage in 1879, who wrote of a special band of muscle around the ducts and ampulla of the cat.

The significance of Oddi's studies, 1887–1888, rests not so much on his anatomical descriptions as upon his physiological studies of the sphincter which bears his name. He was the first to measure the resistance which could be maintained by this muscle in animals, and he attributed to the sphincter the function of retaining bile. Later, in 1894, as a result of his experiments, he claimed for the sphincter a nervous control independent of the neighbouring intestinal muscle. In the same year Doyon clearly stated that the gall-bladder and sphincter constituted a functional unit.

The first histological description of the human sphincter came from Znaniecki, 1895. He observed that the number of transverse fibres of the choledochus increased perceptibly at the level where the duct discharges into the ampulla to a degree constituting a sphincter.

Hendrickson, 1898, published the first study of the human sphincter based on maceration methods.

For the next twenty years few additions of major importance were made and then interest in the subject was again aroused. At this time and for many succeeding years there was no unanimity of opinion as to the nature of the investing musculature surrounding the intramural part of the common bile-duct. One school believed that the whole mechanism was entirely controlled by the strongly acting muscle coat of the intestine which surrounded the duct on its oblique passage through the duodenal wall. Others believed that the physiological and anatomical evidence suggested the presence of an intrinsic choledochal musculature which was capable of independent action, but whose action was often overshadowed by the surrounding and strongly acting muscle of the duodenal wall. The anatomical and physiological contributions of more recent years have not entirely succeeded in clarifying the position, so that there is no complete unanimity of opinion. The advancement in knowledge as a result of the work in these two groups is summarized here.

Anatomical Contributions.—After Hendrickson's work in 1898, further descriptions of

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the choledochal sphincter in man were meagre and scattered until the papers of Nuboer, the Dutch surgeon, in 1931, and more especially those of Schweigler and Boyden, in 1937. These latter two workers approached the problem of the anatomy of the human choledocho-duodenal apparatus from a new angle, for they studied serial sections and reconstructions of human embryos at various stages in development. They were able to demonstrate quite conclusively the existence of a separate sphincter muscle around the intramural part of the common bile-duct. They traced the development of this band of muscle-fibres from mesoderm which appears two weeks later than that from which the circular muscle coat of the duodenum is developed. They described two separate parts of the sphincter—the sphincter choledochus superior, the fibres lying around the extra mural part of the duct—and the sphincter choledochus inferior, previously known as the sphincter of Oddi, lying just distal to the muscular coats of the intestine and continuous with the muscle lining both sides of the ampulla and ending just before the papilla, which they regard as a passive structure. (*Fig.* 365.)

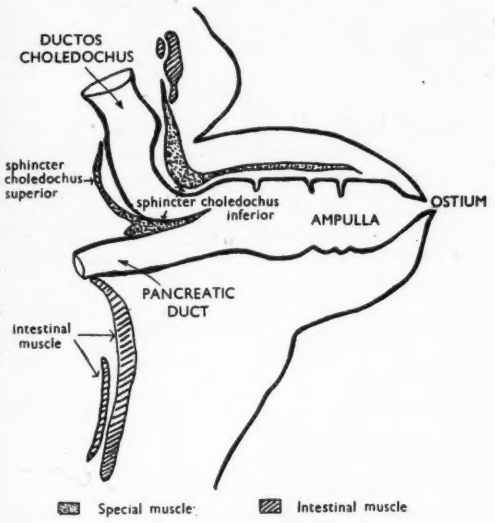

FIG. 365.—Diagram showing the choledocho-duodenal apparatus in the human embryo almost at full term. (Schweigler and Boyden.)

This is a different conception of the anatomy of this part from any previously advanced. For further details, Schweigler and Boyden's paper should be consulted. These workers have done much towards clarifying the structure of the human anatomy of this region. They assume as a result of their studies that the sphincter choledochus inferior is capable of acting independently of the muscle coat of the duodenum; their diagrams explain the relationship of the pancreatic and common bile-ducts, and offer a sound anatomical basis for an explanation of biliary dyskinesia based on spasm of this independent musculature. A study of the accompanying diagram also explains why there is no obstruction of the pancreatic duct during the attacks.

The work which has been done on the anatomy of this region in other species is not so convincing. As the author's own experimental work was performed on dogs, the anatomy of the choledocho-duodenal region in this animal was of particular interest. Unfortunately, the contributions are meagre and opinion is somewhat divided. Thus while Mann and Giordano, 1926-1927, claim the existence of a separate anatomical sphincter in the dog, Halpert, 1932, maintains that there is no true sphincter which is completely independent of the intestinal musculature. Boyden, 1937, notes that although there is a layer of intrinsic muscle ensheathing the duct in the dog this is not so highly developed as in man. The relationship to the intestinal muscle, especially along the upper margin, is much closer, and it would appear likely that the intrinsic muscle around the duct would be overshadowed by the force of contraction of the intestinal musculature. In spite of this division of views, we used the dog as it was the best animal available for experimental work and it was felt that a demonstration of the independent action of the sphincter would be the more convincing, for, if such an action could be demonstrated on the dog, it should be easier to demonstrate it on a patient, since the sphincter was so much better developed and separated in man.

Physiological Contributions.—From a physiological standpoint the work has been more extensive.

As early as 1917 Doyon and Meltzer advanced the theory of reciprocal action of the biliary tract, i.e., that contraction of the gall-bladder and relaxation of the sphincter of the choledochus occurred together. In the succeeding years the physiology of the musculature around the common bile-duct was studied and useful contributions were made by many workers. Of these, Elman and MacMaster, 1926, and Potter and Mann, 1926, working on unanæsthetized dogs with cannulæ in the common bile-duct, studied the normal physiology of the biliary tract. They noticed that the flow of bile into the duodenum was to some extent controlled by the tone and motility of the duodenal muscle, an observation which was supported by the work of Burget, 1926, Berg and Jobling, 1927, and others.

In 1931, Lueth, working in Ivy's laboratory, attempted to record the pressure in the intramural part of the common bile-duct, and at the same time the pressure exerted by the duodenal musculature under various conditions in a series of 75 anæsthetized dogs. He was convinced that the intramural part of the common bile-duct possessed a special sphincteric mechanism which was intimately co-ordinated with duodenal peristalsis and tone, but which was capable of isolated and independent action. Although his results suggested this, he was unable to produce definite proof of his belief.

Ivy, who has written a great amount on the applied physiology of the biliary tract, sums up the existing views in a monograph written in 1934. He believes there is an intrinsic functioning sphincter surrounding the intramural part of the bile-duct, and that this sphincter, together with the gall-bladder, constitutes a physiologically functioning unit. He accepts the theory advanced over twenty years ago by Doyon and Meltzer that the contraction of the gall-bladder and relaxation of the sphincter of the choledochus occur together. To the sphincter itself he attributes at least two functions: (1) By closure to effect the filling of the gall-bladder; (2) To prevent reflux of duodenal material into the common bile-duct.

Ivy realizes from his own experimental work the extreme difficulty of attempting to separate the action of the functioning sphincter from the effects produced by the action of the strongly acting intestinal wall. For this reason he refers to the intramural part of the lower end of the common bile-duct as the choledocho-duodenal mechanism. He believes that a more perfect understanding of this mechanism and its control is essential to a greater comprehension of the normal and pathological biliary tract and more especially to explain conditions of motor dysfunction of the biliary passages.

Contributions related to Biliary Dyskinesia.—Aschoff and Bacmeister, 1909, laid the foundation for the development of the conception of a clinical syndrome dependent on disordered motility of the biliary tract by describing in some detail stasis of the gall-bladder.

Schmieden, 1920, first called attention to patients who suffered typical symptoms of gall-bladder disease and yet at operation presented nothing more abnormal in the biliary tract than a distended gall-bladder. He believed the syndrome was due to a kink somewhere in the biliary apparatus which hindered the outflow of bile and caused distension of the bile-passages. Unfortunately, it was not always possible to demonstrate such a kink.

John Berg, the Swedish surgeon, 1922, suggested that the biliary stasis might be due to a functional disorder of the biliary passages. He based his belief on the observation of hypertrophy of the muscle surrounding the ampulla in these cases. Lyon, writing of non-surgical drainage of the gall-bladder in 1923, supported this theory.

Westphal, 1923, was responsible for the development of the concept of the functional cause of biliary stasis. He formulated a general clinical classification of different types of biliary dyskinesia based on a collection of clinical material and animal experiments. His work was an important addition to the subject at that time, but it is doubtful if it is possible to make such clear-cut differentiation into types of biliary dyskinesia, and his observations have never been confirmed. For further details on this aspect of the subject the reader is referred to the article by Ivy and Sandblom, 1934.

A further contribution on the clinical aspect of biliary dyskinesia and its treatment was presented by Newman in 1933. This was the most comprehensive clinical account to be presented up to that date.

Since 1933 many contributions have come from the United States, and a number have been the result of work on the human subject.

Voegtlin and Ivy, 1933; Kipp, 1936; Waltman Walters, 1936–8; Butsch and McGowan, 1936; Best and Hicken, 1935–8; Doubilet and Colp, 1936–8; are some of the better-known workers. It is impossible to refer to all their work individually, but as a result of their investigations a technique was elaborated whereby a simple apparatus was designed to perfuse a physiological saline solution at a known rate into the human common bile-duct through a rubber T-tube which had been inserted at operation to drain the duct after cholecystectomy. The perfusion pressure was the height of fluid which the choledocho-duodenal mechanism was capable of maintaining in a manometer. By taking regular readings from the manometer changes of pressure in the common bile-duct were observed. It was thus found that the subcutaneous injection of morphia in the patient produced not only tremendous rises of pressure in the common bile-duct, but also reproduced in the patient symptoms of pain and distress in the right hypochondrium sometimes accompanied by nausea. This rise of pressure persisted for several hours and was not affected by the injection of atropine or by any of the antispasmodic drugs.

A temporary, but sharp, full of pressure, accompanied by relief of symptoms, was produced by the inhalation of amyl nitrite. Nitroglycerin in doses of 0·6 mg. (gr. $\frac{1}{100}$) caused a slower, but more lasting, fall of pressure.

The general inferences as a result of these discoveries were that: (1) Spasm of the choledocho-duodenal mechanism might be responsible for the pain of biliary dyskinesia. (2) In cases of biliary colic or pain, morphia is not the best drug to use for therapeutic purposes, even in combination with atropine. The relief of pain is due to the central analgesic action of the drug, but at the same time it causes a rise of pressure and increasing distension in the common bile-duct. This is the condition which is causing the symptoms, and the true aim of treatment should be to relieve and not to increase it. For this reason nitroglycerin is now recommended for the treatment of intractable cases of biliary pain, and more particularly for those patients whose pain persists after cholecystectomy.

THE INVESTIGATION

This work was undertaken in an attempt to answer three questions:—

1. How far do the movements and tone of the duodenum and the pressure maintained by the lower end of the common bile-duct act together?

2. Is there any evidence that the sphincter of the choledochus can contract independently of the duodenum?

3. If there is such evidence, what is the stimulus which makes the sphincter open and close—is it nervous, is it chemical, or is it both?

DESCRIPTION OF THE EXPERIMENTAL WORK

Clearly, any experimental procedures undertaken on patients with a T-tube in the common duct would necessarily be extremely limited, and although we have perfused the common ducts of three patients (*see Fig.* 376), the bulk of the experimental work was performed on dogs.

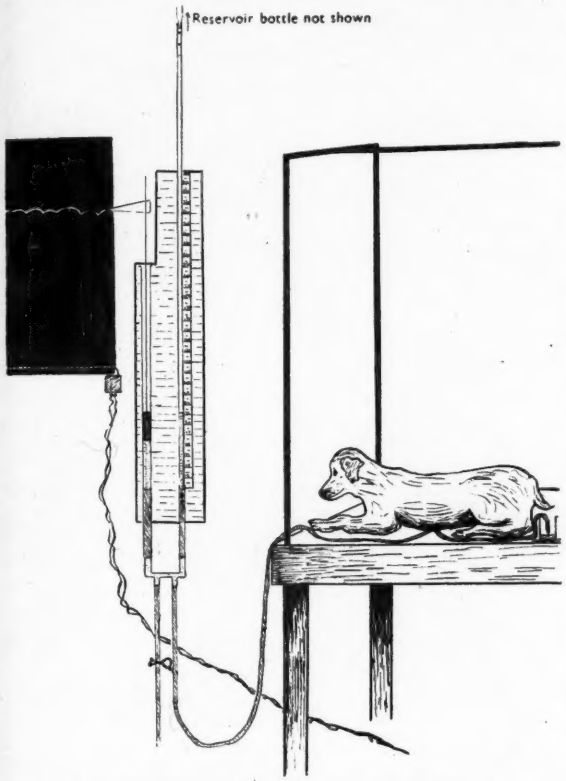

FIG. 366.—Diagram to illustrate the technique of experiments.

A series of 53 dogs was used. Of this total number 40 were done as acute and 13 as chronic experiments. The chronic experiments, in which the common bile-duct of the dog was cannulated under full aseptic precautions and the animal preserved for use for several weeks, were only done in the later stages of the investigation. In all cases large dogs were selected with short coats and wide costal margins to facilitate technical procedures.

For the acute experiments anæsthesia was induced with an ether-chloroform mixture with no pre-operative medication. Anæsthesia was maintained by intravenous chloralose administered in dosage of 0·1 g. per kilo body-weight. Usually this single injection of chloralose was sufficient to ensure anæsthesia for the whole duration of the experiment. It was noted that the experiment did not run so well if more chloralose was given later, and therefore both this and the administration of ether during experimental procedures was avoided if possible.

A simple apparatus was constructed for perfusing fluid through the common bile-duct into the duodenum, and for measuring the resistance offered to the flow of fluid by the choledocho-duodenal mechanism (*Fig.* 366).

From a reservoir bottle, Locke solution flowed down into the narrow limb of a glass U-tube manometer which was mounted just above the level of the experimental table. The rate of outflow of the fluid was regulated to 2 to 3 c.c. per minute. Behind the long narrow limb of the manometer was a measuring scale. The other limb was a shorter tube of a bore wide enough to admit a vulcanite float, the writing point of which recorded on a drum. There were two outlet tubes from the lower arm of the U-tube; one, which was normally kept closed, as an outflow for fluid; one which was connected through a glass junction with a cannula in the common bile-duct. This cannula which was made of glass was inserted through a slit in the wall of the common bile-duct about 1½ in. above its entry into the duodenal wall, and was kept in position by a ligature. The mouth of the cannula pointed towards the duodenum. Another cannula was inserted into the upper part of the common bile-duct with its mouth facing the liver. Through this cannula bile from the upper part of the biliary tract was allowed to drain and collected. The Locke solution thus perfused at a known rate into the duodenum and changes in pressure which were dependent on contraction or relaxation in the choledocho-duodenal region were noted in the measuring manometer tube and recorded by the writing point on the drum.

In some experiments a specially made rubber T-tube was employed instead of the two glass cannulæ and inserted through a slit in the duct wall which was then sewn over it. The cystic duct was in most cases ligatured before the cannulæ were inserted. In a few experiments it was left intact.

The tone and movements of the duodenum were recorded by a condom balloon approximately 2 in. long. The balloon was tied on to the end of a gum-elastic catheter, and was then inserted through a small incision proximal to the pylorus and manipulated through the pylorus to the second part of the duodenum. A ligature was placed in the stomach at the site of introduction to hold it in place. The balloon was connected through the gum-elastic catheter with a water manometer and the manometer in turn attached to a tambour from which a writing point recorded on the drum above the level of the perfusion pressure record. The balloon was inflated with only a small amount of air to prevent any distension of the duodenum.

The blood-pressure was recorded from the right carotid artery to give some indication of the general condition of the animal. In some cases a record of respiration was made, and in a few cases a record through a drop recorder of the outflow of bile from the upper cannula.

The necessary manipulations inside the abdomen were done as quickly as possible with the minimum amount of handling or trauma, and the abdominal wall was then closed with clips and kept closed for the duration of the experiment.

During each experiment a clip was placed on the tube leading to the lower perfusing cannula, but the fluid was allowed to flow from the reservoir at the same rate. The outflow was thus completely occluded, and on the drum was recorded a steady and measured rise of pressure, the slope of which indicated the rate and degree of pressure rise equivalent to a complete closure of the choledocho-duodenal mechanism.

It was thus possible to determine the exact perfusion pressure at any point of the experiment.

Finally, the outflow from the reservoir bottle was estimated so that the amount of fluid entering the system per minute was known.

THE RESULTS

The results, for convenience, are reviewed in the following order :—

1. Record from the Normal Dog.—The perfusion pressure varied in different dogs, but was usually from 130 mm. to 190 mm. Locke solution. It was not maintained at a constant level of pressure, but varied slightly from moment to moment. There were two types of variation—small undulations, probably connected with respiration ; and larger waves, probably related to the changes in tone in the choledocho-duodenal area. The larger variations in pressure, which were of any degree up to 40 mm. or 50 mm. Locke, had a distinct tendency to periodicity—well marked in some dogs, not so well marked in others. In some cases the waves were so regular as to suggest a rhythmic action. In most experiments it was noticeable that rises of pressure were accompanied by increased amplitude of duodenal movements and by increases in the tone of the duodenal wall. Falls in the perfusion pressure were accompanied by diminished duodenal movements and diminution in duodenal tone (*see Fig.* 369). As a general rule when the duodenal movements were noticed to increase, the perfusion pressure began to rise almost immediately.

In experiments where the common duct was cannulated with a rubber T-tube, and in other experiments where although two separate cannulæ were used yet the bile was allowed to flow back into the perfusing fluid, the periodicity of the perfusion pressure was more noticeable than in those experiments where bile was completely excluded by the use of two cannulæ. There was no relationship between duodenal peristalsis and respiratory movement.

From small pieces of records taken at a faster speed, it was noticed that contractions of the duodenum were accompanied by a slight fall of perfusion pressure, while relaxation of the duodenum occurred in conjunction with a slight rise of perfusion pressure.

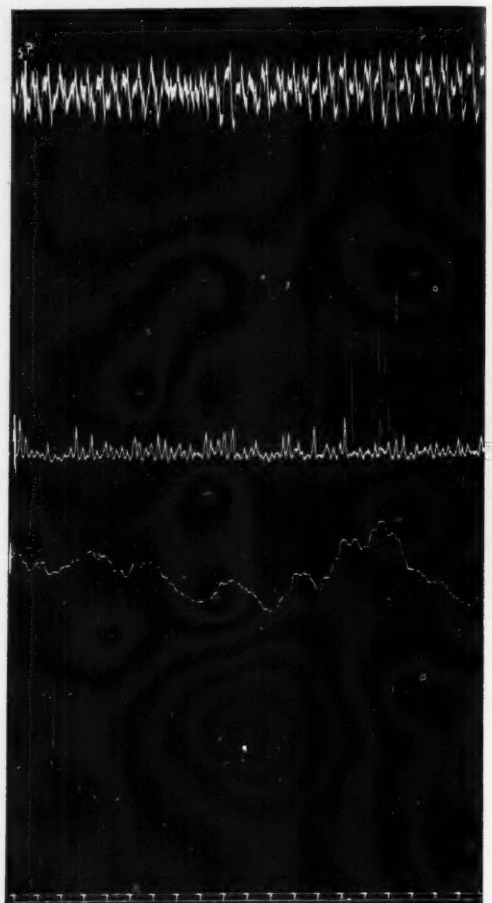

FIG. 367.—Record from normal dog. Slow speed. Common duct cannulated, cystic duct ligatured. B.-p., Blood-pressure; D.M, Duodenal movements; P.P., Perfusion pressure.

In other words, although as a general rule increase in amplitude and tone of the duodenal movements meant rise in perfusion pressure, the perfusing fluid passed into the duodenum during a wave of duodenal contraction (*Figs.* 367, 368).

2. Effect of Morphia and Amyl Nitrite.—Having obtained a clear conception of the appearances of the normal tracing, we determined to examine the effect of subcutaneous injection of morphine on the dog. As this animal can stand large doses of the drug, we began by giving 16 mg. (gr. $\frac{1}{4}$). The effect of the morphine was apparent at periods varying from immediately to ten minutes after injection. The first effect was a great increase in the amplitude of the duodenal

movements and an increase in duodenal tone. The movements increased up to four times their previous excursion. The perfusion pressure began to rise after the increase of intestinal movements. Sometimes the rise began almost simultaneously with the injection, in other experiments there was

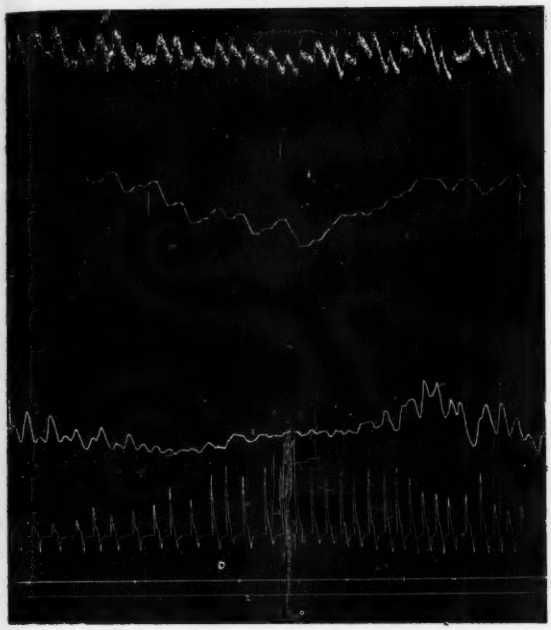

FIG. 368.—Record from normal dog. Fast speed. Connected cannulæ.

a delay of one to three minutes. The pressure rose rapidly to a high level.

Nineteen dogs were treated with subcutaneous injections of morphine in doses varying from 5 mg. to 32 mg. (gr. $\frac{1}{12}$ to $\frac{1}{2}$). In 15 cases there was a rise of pressure and increase in duodenal movements. The perfusion pressure usually rose from about 180 mm. Locke to the uttermost limit of the recording system at 330 mm. In the 4 cases where a marked rise was not obtained, 1 failure was due to faulty technique, 1 was a bad experimental animal, and the other 2 had both had a second dose of chloralose and were very deeply anæsthetized.

To allow recording of the full rises of pressure obtained by morphine injections, a second water U-tube manometer was introduced between the first U-tube manometer and the drum, thus halving the excursion of pressure rises on the drum. Subcutaneous injections of morphine, 16 mg. (gr. $\frac{1}{4}$) then produced a rise of perfusion pressure of from 180 mm. Locke to 600 mm. or more. The rapid rate of the rise of pressure showed that the morphine must have effected complete closure of the choledocho-duodenal mechanism. In one experiment the pressure rose from 150 mm. to over 300 mm. Locke three minutes after injection of only 12 mg. (gr. $\frac{1}{6}$) of morphine.

3. The Effect of Antispasmodic Drugs after Subcutaneous Injection of Morphia.—The subcutaneous injection of atropine in doses up to 1 mg. (gr. $\frac{1}{64}$), had no effect upon the rise of perfusion pressure, and the increased activity of the duodenum after morphia.

The only other antispasmodic preparation used was syntropan, the antispasmodic power of which is very much greater than its other atropine-like effects.

In two experiments, when the usual effects following injection of morphine had been produced, injections of syntropan were given in doses of 10 mg. (gr. $\frac{1}{8}$). In the first experiment when the perfusion pressure stood at 310 mm. Locke, 5 mg. (gr. $\frac{1}{12}$) of syntropan were given intravenously. The pressure rose to 330 mm. and then fell very slightly. This occurred within two minutes. Injection of 4 mg. (gr. $\frac{1}{12}$) of syntropan was then given subcutaneously as the pressure appeared to be rising again. The pressure fell to 220 mm. within the next two minutes and duodenal movements becames lower and smaller. The pressure, however, again rose, and in another ten minutes was back to 265 mm.

In the second experiment subcutaneous syntropan, 10 mg. (gr. $\frac{1}{8}$) was given when the pressure was 290 mm. The pressure rose at first to 330 mm. and then fell slowly until at the end of half an hour it had settled to a constant level of 250 mm. The duodenal movements were slightly diminished.

It was felt that these results were not important enough to justify further experiments along these lines.

4. Effect of Amyl Nitrite after Morphia.—Five of the above series of 19 dogs treated with morphine were given inhalations of amyl nitrite in doses of 30 centimils (ℿv), when the rise of perfusion pressure was at its peak. In all five dogs an almost immediate fall of pressure was produced, varying in extent from 100 to 150 mm. Locke. The fall in the perfusion pressure was accompanied by a slight fall in blood-pressure, but the duodenal movements which had been so grossly increased by the morphine did not show signs of diminution in amplitude. One dog which had a rather high resting perfusion pressure was given an inhalation of amyl nitrite. The pressure immediately fell from 255 to 140 mm. Locke, and the duodenal movements almost ceased, but they tended to recover before the pressure had fallen to its lowest extent. It seems that the amyl nitrite is not powerful enough to combat the effect of the morphine efficiently and the fall of pressure obtained with this drug is but transient. The pressure falls and then begins slowly to rise again to its previous level, while the excursions of the duodenum are not diminished (*Fig.* 370).

5. Effect of giving Sodium Nitrite into the Stomach.—It occurred to us that a weak solution of sodium nitrite given by a gastrostomy tube into the stomach might produce a more satisfactory effect than the transient action of amyl nitrite. In practice the use of this drug would have two advantages: (1) The general effects on patients are not so unpleasant; (2) By passing from the stomach into the duodenum it would directly bathe the duodenal mucosa. In other words it would act directly upon the opening of the common bile-duct into the duodenum, whereas amyl nitrite produces its local action of relieving spasm about the lower end of the common bile-duct only by virtue of its generalized action of relieving muscle spasm.

An experiment was performed by introducing 0·2 g. (gr. 3) sodium nitrite dissolved in about 10 c.c. of water into the proximal part of the stomach through a previously inserted gastrostomy tube, a rise of perfusion pressure first having been produced by subcutaneous injection of morphine 16 mg. (gr. ¼). Within three minutes the fall of pressure was so great and rapid as to produce an almost vertical line upon the drum. The pressure fell from 300 mm. to 80 mm. Locke. At the same time the duodenal movements almost ceased. The duodenum remained

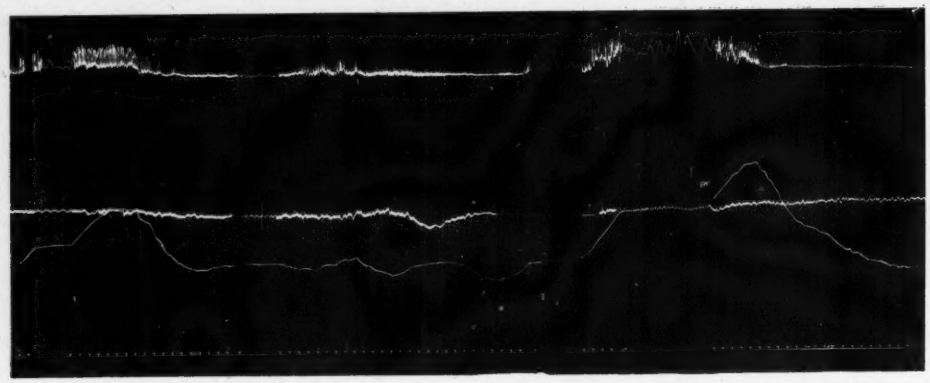

FIG. 369.—Record from normal dog showing periodicity of perfusion pressure and duodenal movements. T-tube in common duct. Excursion halved.

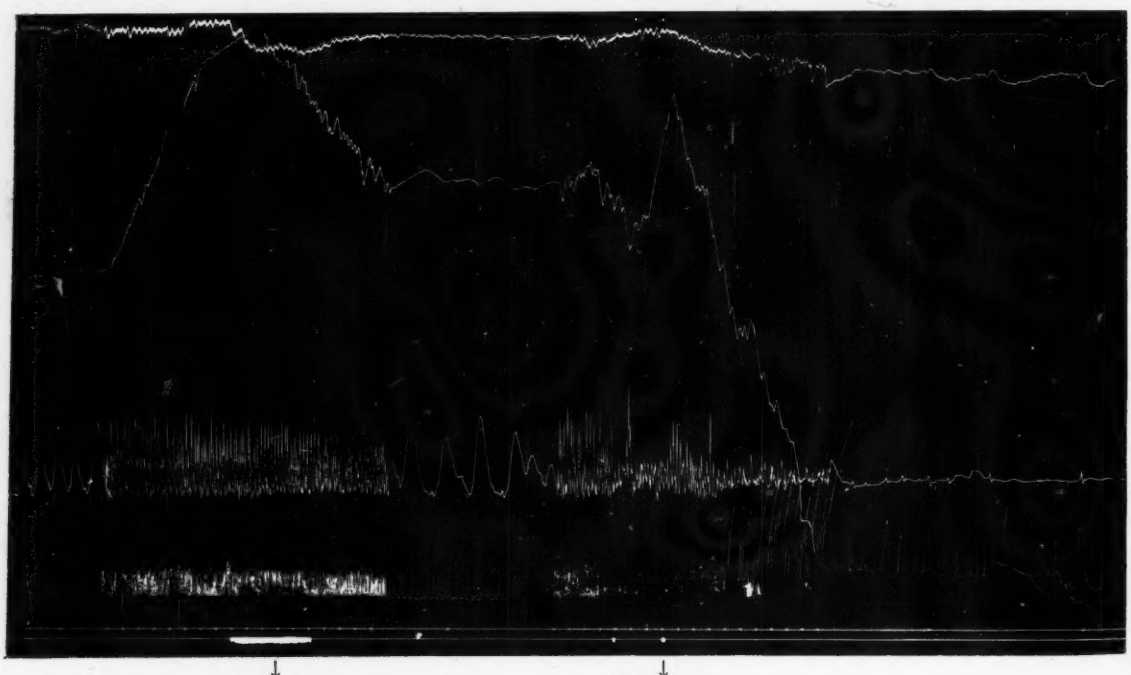

FIG. 370.—Subcutaneous injection of morphia, followed by inhalations of amyl nitrite and then by the introduction of sodium nitrite into the stomach.

quiescent for thirteen minutes before beginning to move again—the perfusion pressure remained at 100 mm. and was not raised even by a second injection of morphia (*Fig.* 370). This experiment was repeated on three other dogs and in each case the introduction of sodium nitrite into the stomach produced a great and lasting fall of perfusion pressure accompanied by extreme diminution of duodenal activity.

This series of experiments had all demonstrated conclusively the close correlation between the perfusion pressure maintained by the choledocho-duodenal mechanism and the movements and tone of the duodenum. It was clearly desirable to develop some method of differentiating the action of the sphincter from that of the duodenum.

A consideration of the last group of experiments suggested that if such a dramatic relaxation of the choledocho-duodenal mechanism could be obtained by the local action of sodium nitrite on the duodenal mucosa, it might be possible to go one step further and to see what would be the effect of introducing small quantities of drugs into the perfusing fluid which bathed the choledochal sphincter on its passage into the duodenum.

To this end a special glass and rubber junction was introduced in the tube leading to the perfusing cannula about 15 in. distal to the cannula itself. Through this special junction injections of the drug were made by means of small all-glass 1-c.c. syringes and No 17 hypodermic needles. Each injection was made up to a volume of 0·1 c.c. or 0·2 c.c. and was introduced slowly and gently so as not to cause any sudden and purely mechanical rise in the perfusion pressure. Great care was taken to exclude the entry of small air bubbles, and every injection of drug was controlled by a previous introduction of an equal volume of the perfusing fluid. By a control experiment, using a dye, it was found that an injection introduced with a syringe at the special junction reached the common bile-duct within a few seconds. It was therefore to be expected that any result from the effect of the injection would be manifested within a very short time.

6. Introduction of Sodium Nitrite into the Perfusing Fluid.—This technique was first employed by introducing sodium nitrite into the perfusing fluid after the perfusion pressure had been raised by an injection of morphine.

In this experiment the pressure rose from a resting level of 130 mm. to 350 mm. Locke following administrations of morphine. The pressure then fell a little and settled at a constant level of 250 mm.; 0·1 c.c. of Locke solution introduced through the rubber junction as described, produced no effect, but the introduction of 10 μg. of sodium nitrite in a volume of 0·1 c.c. immediately following produced an almost immediate fall in the perfusion pressure to 150 mm. Locke. The pressure remained at this level for three minutes, after which it steadily mounted to its previous level of 250 mm. When the pressure was again steady at this level injections of 0·2 c.c. of Locke and 20 μg. of sodium nitrite in 0·2 c.c. Locke were made respectively. The saline produced no effect. The injection of sodium nitrite was followed by a fall of pressure from 250 mm. to 110 mm. Locke. When the pressure had again mounted to the 250 mm. level, repetition of the previous injections produced a fall of pressure to 130 mm. During the injections the amplitude of the duodenal movements showed no diminution (*Fig.* 371).

In a second series of experiments the above technique was repeated using eserine sulphate as a subcutaneous injection and minimal doses of atropine sulphate in the perfusing fluid.

Eserine sulphate, 2 mg. (gr. $\frac{1}{32}$), administered intravenously, produced an immediate and extremely steep rise of perfusion pressure from 130 mm. to 320 mm. Locke. At this stage the choledocho-duodenal mechanism was completely closed. At the same time there was a tremendous increase of duodenal tone with great amplification of duodenal movements. Control injections of Locke solution and quantities of atropine sulphate of 20 μg. were introduced into the perfusing fluid. The first introduction was made when the pressure stood at 320 mm. and was still steadily and rapidly rising. It was noticeable that the slope of pressure rise became not quite so steep. The second injection when the pressure was at 450 mm. was at once followed by considerable diminution of the rate of pressure increase. The third dosage when the pressure stood at 470 mm. was followed by a fall of 450 mm., and the last amount administered at this point was followed by a fall to 420 mm. Locke solution. Throughout this period the tone of the duodenum remained excessively increased as did also the amplitude of the duodenal movements. At this stage in the experiment it became necessary to give the animal intravenous atropine to relieve the extreme condition of spasticity which the eserine had produced. The perfusion pressure and the duodenal movements then returned to their normal level.

The fact that a fall of perfusion pressure could be produced by introduction of minimal quantities of drugs into the perfusing fluid although the amplitude and tone of the duodenal movements remained increased, was the first evidence we had to show that the perfusion pressure and duodenal activity did not always run parallel. The results seemed to yield presumptive evidence of the presence of an independently active sphincter at the lower end of the common bile-duct. It certainly appeared desirable to continue the investigation in the same direction. The further lines of approach were developed with the assumption that if the intrinsic choledochal sphincteric muscle is a true sphincter one should be able to produce upon it pharmacodynamic effects which may be opposite from those produced on the general gut musculature. We decided to

try the effect of adrenaline, which causes a generalized inhibition of peristalsis but a firm contraction of the pyloric and other internal sphincters, and acetylcholine, which, on the contrary, produces stimulation of the gut movements with an accompanying relaxation of the internal sphincters.

It was hoped by the introduction of adrenaline into the fluid perfusing the sphincter to reproduce the effects of adrenergic nerve stimulation. If the choledochal sphincter is a true one such stimulation should result in a diminution of the perfusion pressure made it difficult to be certain of the significance of the results obtained as the effects recorded with such minimal doses were bound to be small. Further, the anæsthesia seemed to depress the sensitivity of the sphincter. Also it soon became evident that it would be necessary to go back to direct recording, and the intermediate U-tube which had been introduced to allow for the great changes in pressure following injections of morphine and eserine was removed. Nevertheless, by conducting several experiments it was found that adrenaline in quantities of 0·1,

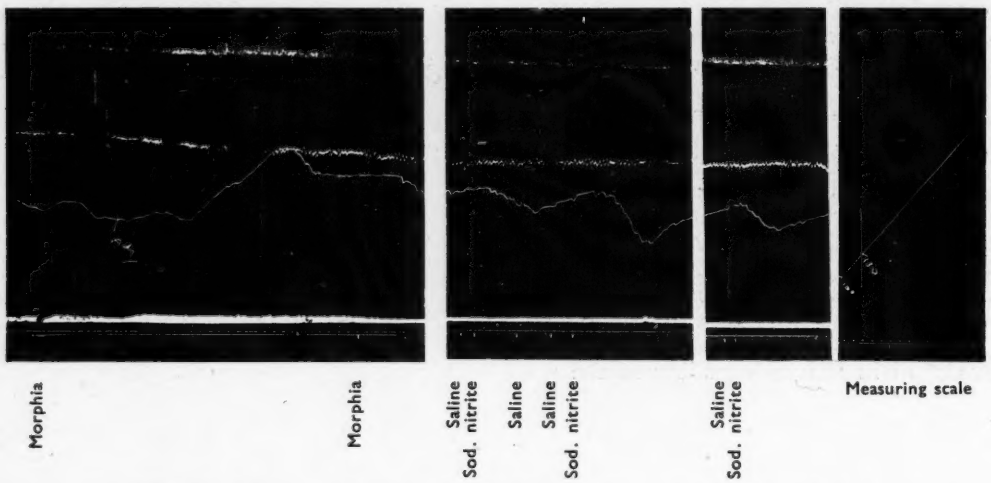

FIG. 371.—Effect of subcutaneous injections of morphine and subsequent action of minimal amounts of sodium nitrite in the perfusing fluid. T-tube in common duct, cystic duct ligatured.

tone and movements of the gut, with an accompanying closure of the sphincter. On the other hand, acetylcholine should reproduce the effects of cholinergic nerve stimulation, and result in an increase in the movements and tone of the duodenum and a relaxation of the sphincter.

By perfusing substances over the sphincter itself such small doses can be used as will not affect the general gut muscle, but will have some action on the sphincter.

With these principles in mind, and with the added hope of finding the stimulus for the action of the sphincter, minimal doses of adrenaline and acetylcholine were employed in the following way.

The preparations were introduced into the perfusing fluid as before. Each dosage was made up to a volume of 0·1 c.c. and each injection was controlled by the introduction of 0·1 c.c. of the perfusing fluid. Standard solutions of adrenaline and acetylcholine were used, diluted immediately before use to the desired strength. The initial strength of the solutions in each case was 0·1 μg. in 0·1 c.c.

7. Effect of introducing Minimal Doses of Acetylcholine and Adrenaline into the Perfusing Fluid.—The experimental results on the anæsthetized animals were rather disappointing. The inherent and periodic changes in the 0·2, and 0·4 μg. caused a slight rise followed by a fall of perfusion pressure, while acetylcholine in the same amounts had the opposite effect, the pressure first falling slightly and then rising. The latter rise of pressure in every case exceeded the previous fall.

These results were promising, for if adrenaline produced an initial rise of pressure this could not be due to any action of adrenaline on the gut muscle. The result might well be interpreted as an initial rise in perfusion pressure caused by the adrenaline stimulating the sphincter, while the later fall in pressure could be caused by the inhibitory action of adrenaline on the intestinal muscle. The converse applies to acetylcholine —the initial fall of pressure might be due to the relaxing action on the sphincter while the later and stronger rise of pressure might well be due to its stimulant action on the gut.

In order to obtain better and more clear-cut results it was decided to repeat these experiments on unanæsthetized dogs.

Effects produced on Unanæsthetized Dogs. —A series of selected dogs were operated on under ether anæsthesia and aseptic precautions. In the first two dogs the common bile-duct was exposed by a laparotomy incision and through a small slit in the wall, a specially made and very small rubber T-tube was inserted. The end of

the tube was brought out through a stab wound in the abdominal wall and the laparotomy incision sutured. The end of the rubber tube was tied off so that while the wound was healing the bile passed into the duodenum in the usual way.

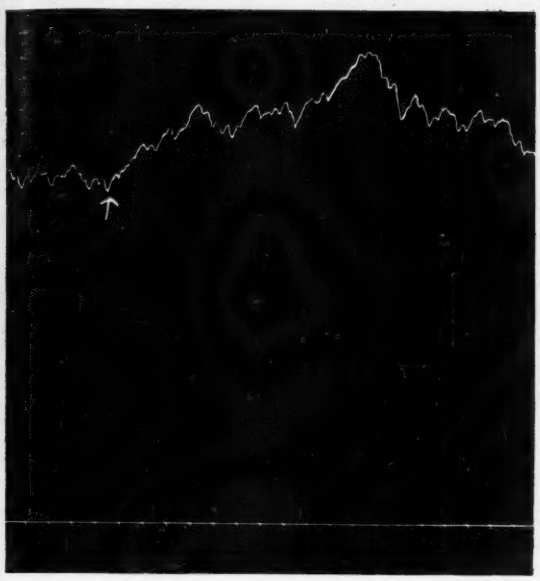

FIG. 372.—Effect of adding 0·2 μg. adrenaline to the perfusing fluid.

Sterile dressings were applied and a special type of many-tailed bandage. Each dog was made to wear a broad brass collar which was padded with cotton-wool and gauze. This made it impossible for them to bite off the bandage and tube in the early days after the operation.

The second series of dogs were operated on as before, but instead of the rubber T-tube two glass cannulæ were introduced and tied into the common bile-duct with silk, one pointing downwards towards the duodenum, and one upwards towards the liver. The two connecting tubes of rubber were brought out through the laparotomy wound and were connected together outside the abdomen with a small sterile piece of glass tubing, so that when an experiment was not in progress the bile could still pass round into the duodenum through the rubber tubing. When performing an experiment the glass tubing was removed and the perfusing apparatus was connected to the tubing of the lower cannula while the bile from the upper cannula was allowed to collect in a glass vessel.

These dogs were maintained in good health for several weeks in this way. Ultimately, in spite of aseptic precautions, they all became infected and developed a cholangitis. It was quite easy to train the animals to lie quietly on the table while they were connected to the perfusing apparatus and the experiment allowed to proceed. They were always given a feed of meat when the experiments were finished.

The use of these unanæsthetized animals had the advantage that the experiment was carried out under more normal conditions. It had the disadvantage that the duodenal balloon could no longer be used, and it was therefore impossible to see what was happening to the gut movements. However, taken in conjunction with the first series of experiments the advantage definitely outweighed the disadvantage.

Our dogs were not always used after a period of prolonged fast as was the method of some previous investigators. As we were using drugs to obtain our effects we saw no particular advantage in always using a fasting animal.

It was noticeable at once that the type of record obtained from these unanæsthetized dogs varied slightly from that previously recorded in the chloralosed animals. The two types of waves which had been previously seen were better shown. In particular the small waves were very clearly demonstrated. Both in the dogs with the T-tube and those with the double cannulæ the resting perfusion pressure was lower than in the anæsthetized animal, varying from 90 mm. to about 120 mm. of Locke. Any marked alteration in body posture resulted in definite changes in the pressure, and for this reason it was desirable to train the dog to lie relatively still.

Fresh solutions of adrenaline and acetylcholine were made up from a standard solution of 1 mg. (gr. $\frac{1}{60}$) per mil just before the introduction into the perfusing fluid. The solutions were arranged so that the strengths varied from 0·1 μg. in 0·1 c.c. to 0·4 μg. in 0·1 c.c.

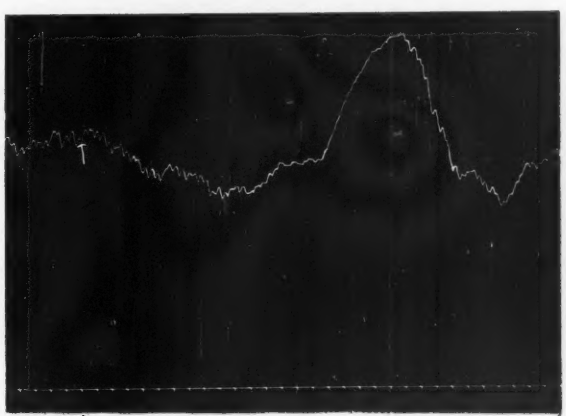

FIG. 373.—Effect of adding 0·2 μg. acetylcholine to the perfusing fluid.

The results obtained by the use of these amounts of adrenaline and acetylcholine in the unanæsthetized animal were much more consistent than those obtained in the acute experiments. Furthermore, there was much greater sensitivity, and variations in pressure were greater in extent. The main difficulty in the technique lay in waiting until the perfusion pressure had reached a fairly

steady level before introducing the drug into the perfusing fluid. If this was not done it was difficult to know whether the rise or fall in pressure was not due to natural variations.

The usual effect obtained with the smaller doses of 0·1 μg. and 0·2 μg. in 0·1 c.c. was that with adrenaline there was an initial slight rise in the perfusion pressure followed by a fall to slightly below the standard level of pressure. With acetylcholine the effect was exactly opposite, the pressure first falling and then rising considerably above the previous resting level. *Figs.* 372 and 373 are the best records which were obtained. The dosage in both cases was 0·2 μg. in 0·1 c.c., and although not shown on the tracing owing to comparator tubes. It was found in a number of experiments that the usual effect of alkalinization of the fluid to a considerable degree, i.e., to a *p*H of 8 or more, caused a fall in the perfusion pressure, whereas acidification to a *p*H of 5 and under caused a rise in the perfusion pressure. Introduction into the perfusing fluid of a dog of its own hepatic bile, collected during the experiment from the upper cannula tube, and alkalinized or acidified in the same manner, caused similar effects. In practice it was found that more N/10 KOH and HCl was necessary to alter the *p*H of the bile as it is a buffered solution. *Fig.* 374 illustrates the variations in pressure obtained by varying the reaction of the bile. It can be

FIG. 374.—Observations on varying reaction of the perfusing fluid by the addition of dog's bile which has been acidified and alkalinized.

limitation of space, both injections were previously controlled by the introduction of 0·1 c.c. of Locke with no effect.

These results seemed definitely indicative of sphincteric effects divorced from effects which were attributable to changes in the general gut musculature, and it seemed desirable to continue the investigation along similar lines with a study of the fluid which normally perfuses the sphincter —namely, the bile itself.

The Effects of introducing Bile into the Perfusing Fluid.—It appeared logical to determine whether the reaction and the composition of the bile itself might not be the stimulus which was responsible for the opening or closing of the sphincter of the choledochus. Since bile is known to be a cholagogue and to stimulate the movements of the gut, it is important to know what would be the effects of varying its composition.

Using the same technique on the unanæsthetized dogs, we introduced into the perfusing fluid Locke solution in volumes of 0·1 c.c., the *p*H of which had been altered by the addition of varying amounts of N/10 potassium hydroxide and N/10 hydrochloric acid. The *p*H of the resulting fluid was estimated approximately by the use of appropriate indicators and standard seen that increasing the amount of acidified bile which was added to the perfusing fluid caused proportionately increasing rises of pressure in the system. It seemed then that variations in the reaction of the bile occurring in a greater degree than that which occurs normally may be a factor in the alterations in the tone of the sphincter choledochus.

The other constituents of the bile which were felt to be worthy of investigation were the bile-salts, more particularly since recent work has centred upon the importance of the bile-salts in disorders of the biliary tract.

Again the same technique was used. The dog's own hepatic bile containing known amounts of sodium taurocholate was added to the perfusing fluid and controlled by the introduction of equal volumes of Locke solution and of hepatic bile. Sodium taurocholate was used as that is the principal bile-salt contained in dog's bile ; 100 c.c. of bile from the normal gall-bladder of a dog contains about 20,000 mg. of sodium taurocholate. It was found that introduction of gall-bladder bile, or of hepatic bile to which sodium taurocholate had been added (in proportion of 1 g. to 5 c.c. of bile, thereby making the concentration of bile-salt stronger than in normal gall-bladder bile) produced a consistent fall of perfusion

pressure, i.e., it caused the sphincter to open (*Fig.* 375.) The fall of pressure thus produced was greater in extent than the variation in pressure produced by the introduction of acetylcholine and adrenaline.

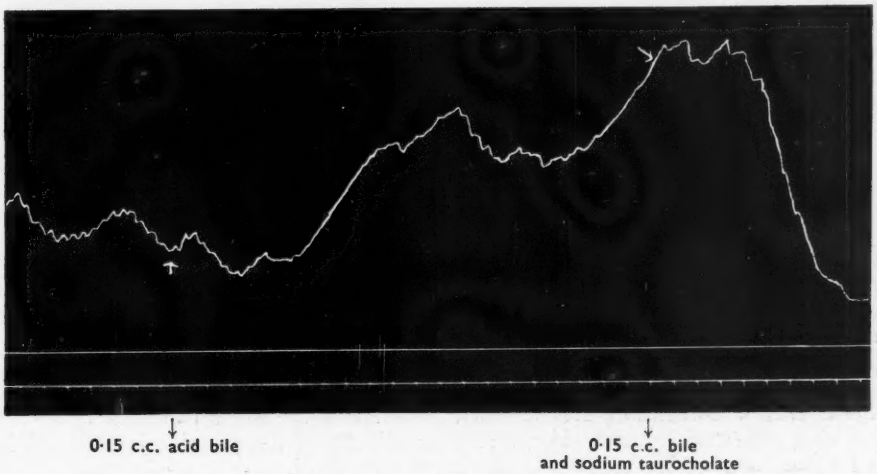

FIG. 375.—Effect of adding acidified bile and then bile plus sodium taurocholate to the perfusing fluid. *p*H of bile = 7·4; *p*H of acidified bile < 6.

In a final series of experiments the effect of subcutaneous morphine injection on the unanæsthetized dogs was investigated. In every case the result was the same. There was an immediate and rapid rise of pressure, the slope of the rise showing that the choledocho-duodenal apparatus was completely contracted. After subcutaneous injection of morphine 10 mg. (gr. $\frac{1}{6}$), the pressure rose from a resting level of 90 mm. to well over 300 mm. of Locke. Two or three minutes after the injection the dog became restless and salivated profusely. These signs were followed by nausea and vomiting, after which the pressure went on rising to its maximum. Even small doses, such as morphine 5 mg. (gr. $\frac{1}{12}$), produced similar symptoms. Concurrently with the rise of pressure the drainage of bile from the upper cannula ceased completely. In one experiment the introduction of the animal's own bile to which sodium taurocholate had been added resulted after each injection in a slight cessation of the rise in pressure, which afterwards continued to rise. It was only after the introduction of 0·95 c.c. of this bile into the perfusing fluid that the pressure slope began to level out at a height of 260 mm. After the further introduction of 0·8 c.c. of the bile-salt in bile when the pressure had reached 300 mm. the pressure began definitely to fall, and in seven minutes had fallen back to 250 mm. and was still falling.

During the course of the experiments on the unanæsthetized dogs certain points of interest presented themselves. As the dog became infected the bile which was collected from the upper cannula became thick and rather cloudy and smelt foul. Concurrently the resting level of the perfusion pressure rose and after the course of a day or two became very high. Thus in one dog where the resting perfusion pressure was at 90–120 mm. at the beginning of the series of experiments, a fortnight after the initial operation, when infection had supervened, the pressure was at 185–200 mm. resting level. The record of the

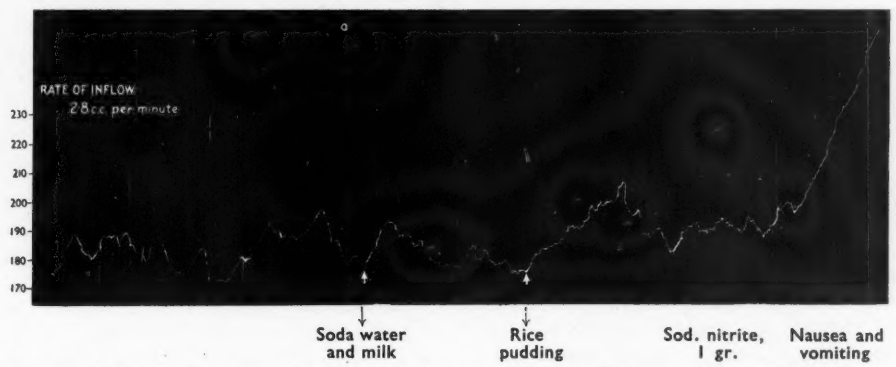

FIG. 376.—Perfusion of common bile-duct of patient two days after cholecystectomy and choledochotomy.

perfusion pressure also altered, the curves seen on the normal tracing becoming flattened. This particularly applied to the small waves. In the initial days of infection when these changes were first noted the animals appeared to be in fairly good health, and did not refuse their extra ration of meat after the experiment.

It was also noticed that when the experiment was begun, if the bile was not draining from the upper cannula, the pressure registered through the lower cannula was higher than usual and remained so until bile began to be discharged from the upper cannula. In some cases where this did not occur readily it was found that administration of meat to the animal caused a free flow of hepatic bile and the pressure then dropped to the normal resting level. On two occasions when the obstruction of a free outflow of bile appeared as if it might be due to viscid gall-bladder bile forming a mechanical obstruction in the upper cannula, 5 to 10 c.c. of saturated sodium bicarbonate was introduced with a syringe into the upper tubing to facilitate solution of the thick mucus. The dog showed almost immediate indications of uneasiness and discomfort which finally resulted in nausea and vomiting. It appeared therefore that a rapid rise of pressure in the upper part of the biliary tract resulted in pain and vomiting.

The apparent connexion in the action of the upper and lower ends of the biliary tract in this way was interesting, more particularly as the two ends were in this case virtually separated by the introduction of two cannulæ tightly tied into the common bile-duct.

DISCUSSION

Examination of the results and records of this work revealed that an answer can be deduced to all three of the questions which we had before us when this investigation was undertaken.

The normal records of the first series of experiments on the unanæsthetized animals show clearly that there is a close relation between the movements and tone of the duodenum and the control of the flow of bile into the intestine. The normal periodic rises of pressure so often seen were invariably accompanied by increase in the tone and amplitude of the duodenal movements while the corresponding decreases in perfusion pressure were concurrent with diminution in the duodenal activities. This was demonstrated independently of whether the common bile-duct was cannulated with a T-tube or with two separate glass cannulæ.

The relationship is no new fact and was demonstrated by Lueth in 1931. Even before this the relationship has been suggested by the observations of Elman and MacMaster, 1926; Berg and Jobling, 1927, Burget, 1926; Giordano and Mann, 1927; and others.

Although Lueth investigated the effects of a series of drugs which he administered intravenously, he did not comment on the results obtained with morphia. The records in the present work which demonstrate the great rises in perfusion pressure following the subcutaneous injection of this drug and the concurrent increases of duodenal tone and motility illustrate most convincingly the relation between the two effects. In every experiment great increases in duodenal activity immediately preceded the rapid rise in the perfusion pressure. Further confirmation is to be found in the records showing the result of the eserine sulphate injection.

Fig. 370, which shows the rise of pressure with morphine and the fall of pressure after sodium nitrite solution had been introduced into the stomach, illustrates well the almost complete cessation of duodenal activity which accompanied the very considerable fall in perfusion pressure. From these records therefore it is certainly possible to confirm and substantiate still further the results of Lueth.

Evidence in favour of the existence of an independent intrinsic intramural sphincter is provided by the experiments in which minimal amounts of drugs were introduced into the perfusing fluid.

The reduction of the high perfusion pressure produced by morphine with the introduction of very small quantities of sodium nitrite illustrates this point. Confirmation is to be found in the records of experiments where high pressures were produced by eserine sulphate injections and later reduced by the introduction of small amounts of atropine into the fluid which bathes the sphincter. In these cases the tone and motility of the duodenum remained at an increased level and variations of the pressure did not go hand in hand with variations in duodenal activity.

These initial observations were confirmed still more convincingly by the series of experiments which employed adrenaline and acetylcholine in unanæsthetized animals. It has already been pointed out, and is here emphasized, that were the sphincter of the choledochus a true sphincter one would expect to be able to produce upon it pharmacodynamic effects opposite from those effects upon the gut musculature.

The records here presented may be interpreted as illustrating this point. In *Fig.* 372 the introduction of 0·2 μg. of adrenaline into the perfusing fluid resulted in an initial and incomplete closure of the sphincter with a resulting rise in perfusion pressure. The later fall of pressure due to relaxation of the sphincter may be explained by the inhibitory effect of the adrenaline on the gut muscle. As was to be expected acetylcholine produced the opposite effect, the same dosage of this substance producing an initial slight fall of perfusion pressure presumably due to relaxation of the sphincter. This was followed quickly by a considerable rise of pressure which was most probably explained by the stimulating effect of acetylcholine on the gut (*Fig.* 373). If there were no intrinsic intramural choledochal sphincter and the perfusion pressure and degree of duodenal

activity went always hand in hand, one would expect the introduction of adrenaline to the perfusing fluid to result in an immediate fall of pressure owing to its inhibitory effects on the gut, and acetylcholine to cause an immediate rise of pressure.

It was noticeable that 0·2 μg. was the optimum dosage for the purposes of our experiment to obtain the best result. Smaller amounts produced similar results in a less-marked degree. Larger amounts produced rather variable results, the duodenal effects then apparently overshadowing any effects produced by the sphincter itself.

While it is not suggested that these experimental results are absolute proof of the fact that the intrinsic choledochal sphincter acts independently of the duodenal musculature in the dog, at the same time it makes it extremely likely that this can in fact occur. Probably the duodenal activity exerts a strong influence on the pressure in the common bile-duct, but variations in pressure independent of the state of the duodenum at the time appear to be possible. Lueth also reached this conclusion, but was unable to produce any real experimental proof of his statement. These results confirm his theory.

If the independent action of such a sphincter can be demonstrated on the dog, where the investigators appear doubtful of the anatomical separation of the intrinsic choledochal sphincter muscle from the circular muscle coat of the intestine, it would apparently be far easier to demonstrate it in the human subject where Schweigler and Boyden's work has shown such complete anatomical and developmental separation.

The conception of a spasm of the independent choledochal sphincter would place the pathology of biliary dyskinesia on a level with other conditions of neuromuscular inco-ordination. The distension of the biliary passages which would result from the spasm of the choledochal sphincter would explain the pain and discomfort of the clinical syndrome. It has been shown in the experimental unanæsthetized animal that distension of the biliary passages and the rise of pressure which occurs in the common bile-duct as a result of morphia causes extreme discomfort and nausea.

The nature of the stimulus which is responsible for the contraction or the relaxation of the choledochal sphincter is not so easily discovered.

The effects obtained by the use of adrenaline and acetylcholine with which we aimed at simulating the effects of stimulation of the sympathetic and para-sympathetic nervous system respectively, were not considered to be sufficiently marked to permit the explanation that the discharge of bile into the duodenum is a purely neuromuscular action.

On this point Ivy (1928) is in agreement, since it was he who postulated the occurrence of cholecystokinin as the stimulating factor causing initial contraction of the gall-bladder to start the flow of bile after a meal.

In reviewing the factors which may be of importance in initiating and continuing the flow of bile into the duodenum, one must consider the composition of the bile itself. If the introduction of various substances into the physiological fluid passing through the choledochal mechanism can influence the resistance it offers to the flow as in our experiments, it is surely logical to suppose that gross alterations in the composition of the normal bile may do the same. It may be that the nervous mechanism is one which only comes into effect when the environmental conditions are suitable—such a condition may well be the composition and reaction of the bile itself.

In the last two years considerate attention has been directed to the composition of the bile and the importance of variations in its constituents in relation to cholecystitis and cholelithiasis—facts first suggested by Rous and MacMaster (1921).

Johnston and Ravdin, 1933, Andrews and his co-workers, 1931–2, Reinhold and co-workers, 1937, and Doubilet and Colp, 1936 and 1938, are a few of the workers who have contributed to the study of the bile. They have all laid stress on the importance of the bile-cholesterol ratio of bile and shown that diminution in the concentration of bile-salts will tend to produce a precipitation of cholesterol. Andrews showed that the quantity of bile-salts was decreased in human gall-bladders with cholesterol stones. This work confirmed the results of Newman, who had done a similar investigation on autopsy material (1931). Doubilet and Colp suggested that the decrease in the bile-salt–cholesterol ratio could be explained in one of two ways: (*a*) By the increased absorption of bile-salts by damaged gall-bladder wall; (*b*) By lowered secretion of bile acids by the liver.

This brief survey indicates that there is a prevalent belief that the bile-salt concentration, more particularly in its relation to cholesterol, is an important factor in the working of the normal and abnormal biliary tract.

Our experiments conducted on the unanæsthetized dog indicate the importance of variation in the *p*H and the bile-salt concentration of the bile. As far as our work was concerned the bile-salt–cholesterol ratio is not important since there is very little cholesterol in dog's bile. The concentration of sodium taurocholate, as the principal constituent of dog's bile, was of importance. The addition of sodium taurocholate to the hepatic bile of the dog introduced into the perfusing fluid definitely caused a relaxation of the choledochal sphincter and a flow of fluid into the duodenum. The approach in our experiments was not a biochemical one, but the evidence is definitely confirmative of the importance of the bile-salt concentration as a factor in initiating the flow into the intestines. The *p*H of the bile also appears to be of some importance. Gross acidification of the bile caused contraction of the choledochal sphincter with increase in the perfusion pressure, while alkalinization of bile

produced relaxation of the sphincter with resultant fall in the perfusion pressure.

Rous and MacMaster, 1921, first announced the concentrating power of the gall-bladder, and it is now known that the human gall-bladder is capable of concentrating the bile from four to ten times, and in the process alters the reaction of the bile from a *p*H of 7·8 to about 6·3, i.e., from slight alkalinity to slight acidification. Also stasis of bile produces increased acidity of bile, as does also infection with *B. coli*.

Since the choledochal sphincter is normally in fasting periods closed to hepatic bile in order to allow the filling of the gall-bladder, it may be that it is also closed to bile which is not of a suitable reaction and concentration.

Considering the experimental results here obtained in conjunction with the many facts about the composition of the bile obtained by other workers quoted in this paper, it is possible to formulate a new theory to explain the symptoms which characterize chronic disease of the gall-bladder and biliary tract.

When the normal person takes a meal there is almost immediate response of the biliary tract. The gall-bladder contracts either as a result of a nervous mechanism, or as a result of the hormone cholecystokinin described and named by Ivy (1928). The contraction of the gall-bladder expels well-concentrated and slightly acid bile rich in bile-salts. This bile reaches the sphincter of the choledochus, which relaxes to allow the discharge into the duodenum of normally concentrated and reacting bile.

When the gall-bladder is chronically diseased there are certain changes in the normal process. In the first place the gall-bladder wall may be stretched and atonic or it may be contracted and fibrosed. Both conditions will affect the power of contraction of the organ. The emptying of the gall-bladder therefore will not be so efficiently performed as in the normal viscus. Secondly, the concentrating power of the inflamed gall-bladder is usually diminished—a fact which is known from radiography with di-salts—and has been further confirmed by the work of Doubilet and Colp and by Andrews, who showed that there is a tendency both for the bile-salts to be absorbed rapidly from the inflamed gall-bladder mucous membrane, and also for hepatitis in the liver, which commonly occurs with gall-stones (Flint, 1930), to affect the production of the bile-salts by the liver. The bile will therefore be poor in its constituent elements, but especially so in bile-salts.

It is probable that the result of the intake of food in such an individual will result in a diminished contraction on the part of the diseased gall-bladder and inefficient expulsion of a bile which is poorly concentrated and may be abnormal in reaction.

This bile reaches the sphincter and owing to the abnormality of its composition the sphincter does not readily relax to permit its passage into the duodenum. Certain effects follow both in the biliary tract and in the duodenum.

In the first place the biliary tract will not have emptied itself completely, and when further bile is secreted by the liver there will be a tendency to stagnation and over-distension of the bile-passages. The tension thus produced will give rise to symptoms of discomfort and possibly pain in the right hypochondrium.

If the retention of bile is considerable the tension may even result in reflex nausea and vomiting.

The digestion of the meal is impaired owing to the poor quality of the bile. The diminished bile-salt concentration will affect particularly the saponification of fats, and excessive fat intake will lead to severe symptoms of disordered digestion.

The remote effects of these phenomena will be a chronic back-pressure of bile in the biliary tract. The stagnation thus induced increases the acidity of the bile still further and renders infection of the gall-bladder more likely. Ultimately the liver will be affected and minor degenerative changes will be responsible for still further decreases in the output of the bile-salts. A vicious circle is thus created which once begun will tend always to get worse as time goes on.

If these views are accepted as a working hypothesis, biliary dyskinesia is merely one manifestation of the earliest stage of these changes. The pain of the condition is due to spasm of the choledochal sphincter which will not allow the strongly contracting gall-bladder to empty itself. The resultant distension of the biliary passages may cause nausea and vomiting. The spasm of the sphincter may be due to a temporary decreased production of bile-salts, which in turn may be dependent on a temporary derangement of the liver, with diminution of output of bile-salts.

It would also be possible for temporary decreases in bile-salt output from liver dysfunction to occur after cholecystectomy has been performed. This again might well lead to distension of the biliary passages, with the usual accompanying symptoms.

The above ideas are put forward as suggestions and not as hard facts. Nevertheless, the theory falls into line with out experimental findings and with the clinical picture which is representative of chronic biliary tract disease.

SUMMARY

This paper begins with a survey of the literature on the anatomy and applied physiology of the common duct at its opening into the duodenum, i.e., the choledocho-duodenal region.

Although it has been shown anatomically by recent work that an independent 'sphincter of Oddi' is demonstrable, yet physiologically the presence of an independently active sphincter has never been shown conclusively. The difficulty has always been to differentiate any pure sphincteric effects from the effects caused by the strongly active duodenal wall.

The first part of this experimental study, undertaken on a series of 53 dogs, confirms the accepted fact that the choledochal sphincter and the muscular coat of the duodenal wall usually contract together.

The second part of the study demonstrates that the choledochal sphincter can act independently of the activity of the duodenal wall. This has never before been demonstrated conclusively from a physiological standpoint.

The third part of the study attempts to discover the stimulus which may be responsible for this independent action of the sphincter. The importance of the *p*H of the bile and the concentration of the bile-salts in relation to the working of the choledochal sphincter is demonstrated.

In the discussion, the experimental findings are correlated with the clinical syndromes associated with the pathology of the biliary tract, with special reference to the aetiology of biliary dyskinesia.

I should like to express my thanks to Professor E. R. Flint for suggesting the original subject of this work, and to Dr. W. A. Bain for his advice and help during the course of the experimental procedures.

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SHORT NOTES OF RARE OR OBSCURE CASES

THE SURGICAL AFFECTIONS OF THE APPENDICES EPIPLOICÆ

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LESIONS of the appendices epiploicæ are interesting on account of their rarity, and as such lesions often cause an 'acute abdomen' these cases raise interesting problems in regard to diagnosis. Indeed, a pre-operative diagnosis of a lesion of an appendix epiploica has not yet been recorded. A sufficient number of cases have now been put on record to permit of a classification of these lesions and of a recognition of the clinical syndromes produced thereby. The most commonly encountered lesion is that of torsion of an appendix epiploica. Fiske (1936) collected 42 cases of intra-abdominal torsion of epiploic appendages to which he added 2 cases of his own, and at the present date (1941) the number of cases of primary lesion of appendices epiploicæ is 62. The cause of these torsions is still an undecided problem. The most likely cause of such torsions would appear to be irregular movements in adjacent abdominal viscera, most often neighbouring coils of intestine, and such torsions would be most likely to occur in the case of large appendices epiploicæ with narrow pedicles. It is possible that unusual or exaggerated movements of the abdominal walls might also cause such torsions, but it is unusual to obtain a history of such movements. The effect of torsion of an appendix epiploica is a strangulation of the vascular supply to the appendage causing venous engorgement, extravasation of blood into its fat, and swelling of the appendage. In the more severe grades of torsion gangrene of the appendage would supervene. Pines, Rabinovitch, and Biller (1941) made a study of the blood-supply to these appendages. The blood-vessels of the colon encircle the bowel wall in the subperitoneal layer giving off branches to the bowel walls as they do so. The vessels then run into the fat of the appendices epiploicæ as a U-shaped loop from which branches run proximally into the bowel and distally into the epiploic fat. The main vessels then return to the subperitoneal layer and finally enter the muscle wall near the tinea coli. This arrangement of the vessels in a U-shaped loop facilitates their strangulation when the appendix epiploica is twisted.

Another possible lesion of these appendices is that of infarction. Rabinovitch, Pines, and Biller (1941) draw attention to two of their cases in which the appendix epiploica was not twisted at the time that the lesion was exposed on the operating table. Rabinovitch suggests that engorgement of the venous loop going to an appendix epiploica would be more marked in the distal portion of the loop, and that slight traction of such an engorged loop might elongate and obliterate the lumen of the vein as it enters the appendage and so cause a thrombosis. Once a thrombosis had occurred the changes in the appendix epiploica would be similar to that produced by torsion. While it is impossible to deny that infarction might occasionally occur, it is undoubted that in the vast majority of cases the lesion is one of torsion, and even if no torsion was seen at the time of operation it is possible that the torsion has become undone at some subsequent time. The main symptom produced by torsions of appendices epiploicæ is that of abdominal pain. The site of the pain would vary with the site of the appendix epiploica. Torsion of an appendix epiploica related to the cæcum would produce acute pain in the right iliac fossa, and would closely simulate an acute appendicitis. Pain in the left lower abdomen would be caused by torsions of appendices epiploicæ in the sigmoid colon, and such pains would be mistaken for cases of diverticulitis. Torsion of an appendix epiploica in the transverse colon would cause pain in the neighbourhood of the umbilicus. Associated with the pain is deep tenderness, but abdominal rigidity is usually not evident in these cases. Cutaneous hyperæsthesia has, however, been noted in many cases. Associated with the abdominal pain there might be nausea, vomiting, and low fever. In some cases, diarrhœa or constipation, dysuria, and frequency of micturition have been noted. The leucocyte count is usually only moderately raised in these cases. At the operation, on opening the peritoneal cavity a serosanguinous exudate may be seen. On exploration the twisted plum-coloured epiploic appendage is found, and is readily removed. Should these cases not be operated on it is possible that spontaneous separation of the appendices epiploicæ might occur. Klingenstein (1924) collected 12 cases of loose intra-abdominal bodies which were probably caused in this manner. Occasionally a localized abscess or a fatal peritonitis might occur.

Appendices epiploicæ have given rise to acute intestinal obstruction. Two appendices epiploicæ becoming adherent across the antimesenteric border of the bowel might cause an obstructing constriction. Klingenstein in 1924 reported 3 cases of this complication. Patterson (1933) added

2 more such cases, and McIver (1933) reported a case in which an appendix epiploica had become adherent and had acted as an obstructing band. Baumeister, Hargens, and Morsman (1938) added another case in which three pairs of appendices epiploicæ becoming adherent across the antimesenteric border of the colon had produced obstruction at three places.

CASE REPORT

Torsion of an Appendix Epiploica closely simulating Acute Appendicitis.—

HISTORY.—L. T., aged 27, male, single, English, gunner in the Royal Artillery, was admitted to the Memorial Ward on Feb. 18, 1940, on account of acute abdominal pain. The pain had started two days previously. At first it was felt in the pit of the stomach; it then shifted downwards along the left side of the abdomen, and finally settled in the right lower abdomen. The patient had not vomited and he had felt no nausea. His bowels had been opened five times on the day of onset of the pain, and once on the following day.

ON EXAMINATION.—The patient was lying quietly in bed, his pulse was 72, and of good volume, his temperature 99°. The abdomen was soft except in the right lower abdomen, where it was rigid and exquisitely tender. There was no tenderness in the right loin and no cutaneous hyperæsthesia. It was considered that the patient had acute appendicitis, and he was operated on at 1 p.m. the same day.

OPERATION.—A MacBurney incision was made. The extraperitoneal fat was œdematous. On opening the peritoneal cavity there was seen a plum-coloured organ, with a sharp fissured anterior edge, closely resembling the appearances of a normal spleen. The plum-covered mass was felt to extend downwards

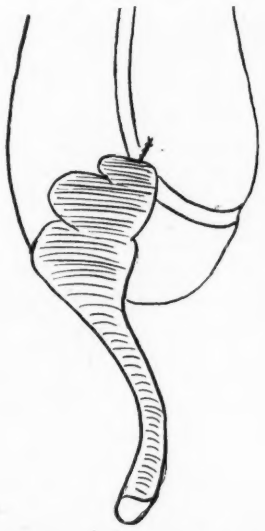

FIG. 377.—Diagrammatic representation of the operative findings. (For description, *see* text.)

towards the pelvis, and it was delivered through the abdominal wound. It was now noted that the plum-coloured organ was about 6 in. long, and that the terminal 4 in. was cylindrical, the diameter of a fountain pen, and in appearance not unlike a distended congested appendix. The tip was, however, yellow. The mass had no mesentery, and at its base there was observed a small pedicle, diameter No. 1 catgut, length 4 mm., tightly twisted at its origin from the side of the anterior tænea coli of the cæcum (*Fig.* 377). The mass was believed to be a deeply congested appendix with gangrene at the tip. This pedicle was ligatured and the mass removed. The wound was closed.

THE SPECIMEN.—It was plum-coloured from extravasated blood. Incision into it showed that it

FIG. 378.—Photograph of the specimen.

had no lumen, and that blood had extravasated into its entire extent except for the terminal ¼ in. at the tip. It was now clear that what had been removed was a greatly enlarged appendix epiploica arising near the anterior tænea coli of the cæcum, which had undergone severe torsion at its narrow pedicle (*Fig.* 378).

Microscopical Report (127/H.40).—The section shows fibrous tissue with large numbers of blood-vessels and free hæmorrhage. There is a certain degree of mucoid change. (W. A. E. Karunaratne.)

These histological findings are unusual, as no fat was seen in the section. A further section was made of the yellow tip of the appendage which to the naked eye appeared to be fat. This, however, on histological examination showed fibrous tissue with no fat.

The very narrow pedicle in this case may have impaired the blood-supply for a considerable period prior to the final torsion, and it is possible that this had caused replacement of the fat by fibrous tissue, and that numerous blood-vessels had formed in this tissue.

The patient made an uninterrupted recovery and left hospital on March 7, 1940.

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A CASE OF BRANCHIAL FISTULA

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The recording of this case has been prompted by the relative rarity of the occurrence of complete branchial fistula in adults.

CASE REPORT

History.—T. C., aged 29, an aircraftman, was seen at the surgical out-patients' department of an R.A.F. hospital complaining of a discharge from the

Fig. 379.—Antero-posterior radiograph of the fistula after injection of lipiodol.

right side of his neck which had been present for 18 months. He had never had any pain, though occasionally he felt a tightness in his throat and sometimes the right side of neck became slightly swollen. He complained of no other symptoms and the irritation of the continued discharge had caused him to report sick.

On Examination.—Average physique. No obvious malformations were seen. Neck: At first sight there appeared to be no sinus visible in the right side, but on pressure over the lower third of the sternomastoid a bead of pus appeared just above and internal to the right sternoclavicular joint. On very close inspection a minute opening could be seen, out of which pus could be made to flow: there was no evidence of any granulation tissue at this opening and the squamous epithelium of the skin was continued into the beginning of the fistula without interruption. No other physical signs were present in the neck.

Other systems: Nothing abnormal found.

X-ray Examination.—A radiograph was taken to determine the extent of the fistula. A blunt lachrymal needle was used and lipiodol was injected into the track. As the accompanying photograph shows (*Fig.* 379) the fistula began as a narrow channel, widening into a fusiform cavity, and narrowing again to its point of entrance above the angle of the mandible in the oral pharyngeal wall (A).

That the fistula was complete is proved by the presence of lipiodol in the pharynx itself (B).

Operation.—This was performed by G./Capt. Stanford Cade, who passed a probe into the fistula and made a small elliptical incision to include the opening. The track was then gradually dissected out, the incision being enlarged as required, its final extent being from the original opening to the angle of the mandible. The fistula was found to pass deep to the sternomastoid at approximately the junction of its middle and lower thirds, thence between the two carotid arteries, and deep to the hypoglossal nerve, to the lateral wall of the pharynx in the region of the supratonsillar fossa. The last ½ to 1 in. of the track was very attenuated and was ligatured with fine silk. The wound was sutured with interrupted horsehair stitches after hæmostasis had been secured, and a corrugated rubber drain was inserted for the first 24 hours. During the dissection it was observed that the whole of the fistula was surrounded by a layer of muscle.

Subsequent Progress.—The patient's post-operative course was uneventful. The drain was removed in 24 hours, the stitches were removed on the seventh day, and he was allowed up on the ninth day. In 14 days the scar was perfectly healed, as shown in the accompanying photograph (*Fig.* 380) and he was allowed to shave.

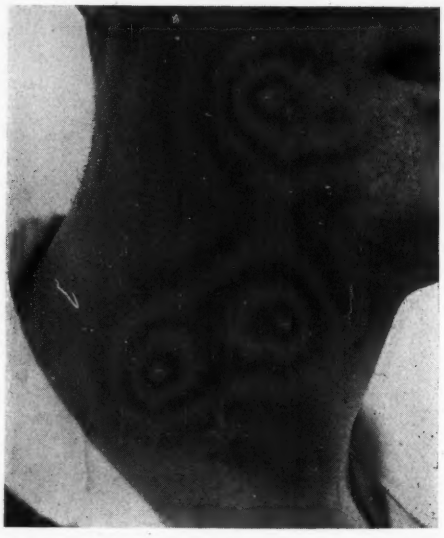

Fig. 380.—Photograph of the patient after operation.

A pre-operative photograph was not taken since the external opening of the fistula was so small that it would not have been demonstrable.

Microscopical Examination of Specimen.—The excised fistula was sent for section (*Fig.* 381). It proved to be a tube of narrow calibre imperfectly lined with ciliated columnar epithelium having masses

of lymphoid tissue in the sinus wall and longitudinally running bundles of voluntary muscle attached to its external aspect. The lining epithelium showed some attempt at mucous gland formation. The typical microscopical appearance of a branchial fistula.

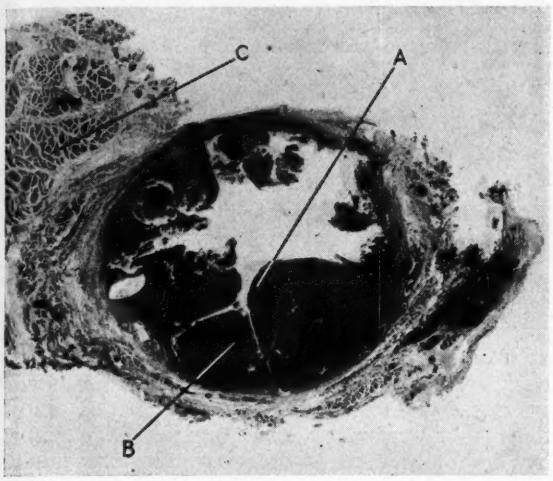

FIG. 381.—Microphotograph of section of branchial fistula. A, Columnar epithelium; B, Lymphoid tissue; C, Voluntary muscle.

DISCUSSION

The aetiology of branchial fistulæ and cysts is said to be a failure of closure on the part of the third, and sometimes the second, cleft in the embryonic pharyngeal wall. There is some divergence of opinion amongst the authorities as to which of these clefts is most commonly involved, Romanis and Mitchiner (1934) state that it is the third cleft and Boyd (1932) states that it is the second.

Weglowski (1908) has suggested that these fistulæ are not cleft remnants at all, but are vestiges of the thymo-lymphatic trunks which are drawn out when the lobes of the thymus descend from their points of origin in the ventral angles of the third clefts. This theory has found little support in the English text-books although it is based on strong anatomical and microscopical evidence, derived from the study both of embryos and adult bodies.

Microscopically (Boyd, 1932) such vestiges are found to be divisible into two types, one lined by squamous cells, and the other by columnar; further, it is observed (Boyd, 1932) that when carcinoma arises in these fistulæ it is of the squamous-cell type. On this evidence alone it is, therefore, impossible to be dogmatic about the theory of origin, but it is within the realms of possibility that both may be right and that such fistulæ may arise in some cases from an unclosed cleft and in others from a remnant of the downgrowth of the thymus gland.

Branchiogenic carcinoma is uncommon, and Wheeler (1938) states that when it does occur it is usually in a male patient and in relation to an unclosed second cleft. The fact that a certain percentage of these fistulæ and cysts do undergo malignant change makes early removal a necessity. In cases where a large cyst is present, the patient is usually eager to have the deformity removed, but in the variety described above, with an almost invisible opening, the size of the scar necessary for radical excision sometimes causes the patient to hesitate before giving his consent to operation.

I am indebted to W./Cmdr. I. MacKay for the histology and to S./Ldr. R. F. Wynroe for the X-ray plate and photograph.

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COMPLETE BLOCKAGE OF THE PULMONARY BLOOD-SUPPLY OCCURRING DURING COMMON ILIAC EMBOLECTOMY

BY G. G. CROWE
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CASE REPORT

A LABOURER, aged 51 years, was admitted to hospital on July 1, 1941, complaining of shortness of breath and pains in all joints since November, 1940.

An electrocardiogram showed auricular fibrillation and arborization block.

At 7.35 p.m. on July 7 the patient complained of sudden pain in the right ankle-joint. At 7.45 p.m. he complained of agonizing pain in the right lower limb. He was sweating, but not markedly collapsed. He could move his toes and there was only slight limitation of the movement of his right lower limb. There was no loss of sensation. Seen at 8.30 p.m., the limb was cold and pale. No popliteal or femoral pulse was present on the right side and no pulse could be felt above Poupart's ligament. There was only 20° of flexion possible at the hip-joint and 50° of flexion at the knee-joint. A left femoral pulse was present. The diagnosis of right common iliac embolism was made. In view of his cardiac condition it was thought that he would be unfit to stand a general or spinal anæsthetic and it was decided to try retrograde removal by exposure of the femoral artery under local anæsthesia.

OPERATION.—Exploration was begun under local anæsthesia at 9.15 p.m., 1 hour 40 minutes after embolism had occurred. Morphia, ¼ gr. and atropine,

$\frac{1}{100}$ gr., were given at 8.55 p.m. The right femoral artery was exposed by mobilizing the sartorius and was found to be pulseless. After clearing the adventitia a small incision was made longitudinally in the arterial wall, the artery above and below the incision being controlled by tension bands of thin rubber tubing. By the use of a ureteric catheter passed up the artery a mass was felt obstructing the bifurcation of the right common iliac artery. Retrograde probing with the ureteric catheter in an effort to dislodge the embolus was unsuccessful, and therefore it was decided to try and milk the clot down from the common iliac artery. With local anæsthesia Astley Cooper's approach to the common iliac artery was carried out. Before attempting to push back the peritoneum, at this stage gas and oxygen anæsthesia was commenced, as the patient complained of pain from the manœuvres in the new incision. The peritoneum was reflected back. A large clot was then felt with the fingers occluding the bifurcation of the common iliac artery and above this the common iliac was pulsating normally. The clot was then broken up and milked down, from the common iliac artery to the incision in the femoral artery, the femoral artery below the incision being controlled meanwhile by the rubber tension band. In all eight pieces were milked down and removed, and after this blood spurted freely from the wound in the femoral artery. This was controlled by the rubber tension band. The distal band was then slackened, the upper held firm, and the collateral circulation proved to be satisfactory. The bands were then held firm, a fine needle with Chinese silk 0000 was introduced into the arterial wall and suture of the wall begun. At this point the patient became suddenly very cyanosed and respiration ceased. The mask was removed from his face and oxygen was administered, but there was no improvement in his condition apart from two small shallow sighing respiratory excursions. The bands were removed from the femoral artery and no pulsation was present in the vessel. The common iliac was palpated, but was not pulsating.

POST-MORTEM FINDINGS.—The *heart* had the appearance of an old rheumatic heart, both sides being enlarged and fibrotic changes present in the myocardium. An old pericarditis with fibrinous adhesions was present. The left ventricle was full of blood. The left auricle contained thrombi. The right auricle was filled with thrombi. The right ventricle was empty but impacted against the cardiac side of the pulmonary valve was a fresh embolus completely occluding the pulmonary orifice. The coronary arteries were narrowed and showed extensive atheroma and sclerosis. The myocardium was of poor quality from fibrosis probably arising from the coronary arteriosclerosis, giving the appearance of interstitial myocarditis. A pleural effusion was present on each side. The largest piece of embolus from the common iliac artery after fixation measured 2 cm. × 1 cm. Microscopically it showed thrombus of a type richly infiltrated with polymorphs as a prelude to organization. The pulmonary embolus was composed of recent thrombus much younger than the common iliac embolus. After fixation it measured 1·8 cm × 0·6 cm.

DISCUSSION

Arterial embolism usually occurs in sufferers from heart disease and with some form of auricular stasis. Further embolic phenomena occurring during the treatment of arterial embolism frequently is the final cause of death. In 7 cases of arterial embolectomy described by Jefferson[1] subsequent embolism occurred in 3 and was the cause of death in 2 cases, 1 due to aortic and 1 due to intestinal embolism. Koucky, Beck, and Hoffman[2] describe 25 cases of peripheral arterial embolism. In 3 of these cases embolectomy was performed, the others being treated by amputation or conservatively. Subsequent pulmonary embolism caused death in 2 of these 3 cases, one 10 hours and one 42 hours after operation. In the case described complete blockage of the pulmonary blood-supply by an embolus occluding the pulmonary valve occurred during operation after an embolus had been removed from the right common iliac artery.

Trendelenburg's operation for pulmonary embolism would have been impossible in this case without damage to the pulmonary valve.

An interesting point was the complaint of pain in the right ankle-joint. This bears out again the fact that the pain in arterial embolism bears no exact anatomical relationship to the level of the obstruction, e.g., impaction in the common femoral artery frequently gives rise to severe pain in the knee-joint.

I wish to thank Dr. A. J. Wright and Dr. J. L. Berry for the post-mortem report.

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REVIEWS AND NOTICES OF BOOKS

The Doctors Mayo. By HELEN CLAPESATTLE. $8\frac{7}{8} \times 6$ in. Pp. 822 + xiv, with many illustrations. 1941. Minneapolis: The University of Minnesota Press. $3.75.

THE story of the Doctors Mayo is one of absorbing interest whether it is regarded as a history of three remarkable men, of the early development of American Institutions, or the growth of medical science over the period of a hundred years.

The present work is no haphazard affair, but the outcome of a considered policy which came about as the result of a conference between the Authorities of the University of Minnesota and the Mayo brothers, on board the yacht belonging to the latter in 1936. The decision to make the publication was forced upon them by the appearance of various unauthorized publications and was entrusted to Helen Clapesattle, herself an historian trained by Stanford Ford, the Professor of History and President of the University of Minnesota.

The author has not only had placed at her service all the documents and staff of the Mayo Clinic, but she has also given five years of unwearied research into the very many factors which contributed to the growth and development of a unique institution. The sources of her information are given in the 82 pages of bibliographical notes which conclude the volume.

The story opens with an account of the life and struggles of the " old doctor " William Worrall Mayo, who was born in Eccles, near Manchester, in 1819. His early education, begun in England and finished in America, his struggles for existence, and his completion of his medical training, give a good idea of the rough-and-ready preparation of one who lived to endear himself to many thousands of patients and to bring up two of the world's most famous surgeons. The chapter entitled the " horse and buggy doctor " gives a graphic account of the strenuous life of a medical practitioner in a pioneer state. Epidemics, the civil war, and the rising of the Indians are all vividly described. He lived to be over ninety and was able to see his sons become famous. How these sons became educated and qualified gives a remarkable picture of early medical education in America and of the triumph of hard work over all the difficulties that they encountered. A notable milestone on their road of progress was the foundation of the St. Mary's Hospital by the Sisters of St. Francis. This Institution, many times enlarged and modernized, continues to this day as the scene of the clinical activities of their successors. It was not until many years later that the Mayo Clinic was founded to accommodate the team of medical men who slowly evolved the idea of co-operative work in diagnosis and treatment. This too has grown and developed until the modern day, in which it physically and spiritually dominates the whole town of Rochester. It is now a building of fifteen stories, besides the surmounting bell tower with four stories, containing the carillion of twenty-three bells. The history of every phase of surgical practice is given with a great wealth of detail. Anæsthesia, asepsis, ovariotomy, appendicectomy, gastro-enterostomy, and many other subjects of surgical speciality are described as these subjects first came before the Mayos and were later developed by them and their colleagues. It is impossible in the compass of a short review to do justice to this most absorbing book, but two strong impressions are left on one's mind; the first is the wonderful capacity that the Mayo brothers showed for a loving co-operation with each other and with all who lived to work with them; and the other is the amazing generosity which they showed in expending the vast fortune which passed through their hands, which they always treated as a trust to be administered for the profession and the community of which they were members.

The only criticism of the book we would venture to make is that the illustrations would be much better if they were distributed about the volume instead of grouped in four sections as is now done.

Architectural Principles in Arthrodesis. By H. A. BRITTAIN, M.A., M.Ch., F.R.C.S., Senior Orthopædic Surgeon, Norfolk and Norwich Hospital and Jenny Lind Hospital for Children; Hunterian Professor, Royal College of Surgeons, England, 1940–41, etc. With a Foreword by HARRY PLATT, M.D., M.S., F.R.C.S. (Hon.), Professor of Orthopædic Surgery, University of Manchester; Honorary Orthopædic Surgeon, Manchester Royal Infirmary; etc. $10\frac{3}{4} \times 8\frac{3}{4}$ in. Pp. 132 + xii, with 144 illustrations, a number in colour. 1942. Edinburgh: E. & S. Livingstone. 21s. net.

IN many ways this is a noteworthy book. In a small compass the author has contrived to give a great number of original ideas and to support them with sound arguments; and then in these times of trouble it is a great achievement to have produced such a well-written and beautifully illustrated monograph.

The author's main idea is that of fixing the joint by means of a bone-graft, so placed as to bear the stress imposed upon it, according to the principles taught by the use of a strut in architecture. This idea was first evolved in the case of the hip-joint, in which a tibial graft is made to unite the femur with the ischium. This is the idea of Trumble's operation, but the graft is placed across the femoral shaft after dividing the latter by an osteotomy. Having satisfied himself of the soundness of this operation and its architectural principles, he has gone on to deal with each joint of the body and has devised methods in each of placing the bone-graft in such a way as to produce the maximum efficiency. In the knee-joint he uses two grafts which cross one another obliquely across the centre of the joint. In the ankle a graft is thrust downwards from the tibia into the astragalus; in the spine use is made of many chip grafts. The shoulder-joint is fixed by means of a specially shaped strut graft from the axillary border of the scapula to the neck of the humerus. Each chapter is accompanied by beautifully drawn illustrations, some of

which are in colour, and is followed by a series of radiographs illustrating actual cases.

We frankly give our whole-hearted admiration to the author for his ingenuity and the way in which he has carried out the principles that he has laid down, but we cannot help an uneasy fear lest the simplicity of surgical procedure has been rather sacrificed in carrying out "architectural principles". At any rate the book will stimulate thought and promote emulation.

Operative Surgery. Including Anæsthesia, Pre- and Post-operative Treatment, Principles of Surgical Technic, Blood Transfusion, and Abdominal Surgery. Edited by FREDERIC W. BANCROFT, A.B., M.D., F.A.C.S., Associate Clinical Professor of Surgery, Columbia University, etc. $9\frac{5}{8} \times 6\frac{1}{2}$ in. Pp. 1102 + xx, with numerous illustrations. 1941. London and New York: D. Appleton-Century Co. 55s. net.

THIS comprehensive volume of well over a thousand pages has many useful and somewhat novel features in its make-up. The basic main facts of operative surgery are rightly stressed and illustrated; much space is given to blood transfusion and pre- and post-operative treatment which is entirely lacking in many books on operative surgery.

Perhaps the most useful section in the book is that devoted to the subject of dieting after surgical operations; this contains a wealth of real practical information which will be of immense value to surgeons. Too often the question of post-operative diet is left to the house surgeon or the sister.

The illustrations on the whole are good; some are excellent, while others are poor; it is a pity that so many illustrations have been used from surgical articles published as far back as 1933, and these are quite dated.

Surgeons will find very sound advice on all the major surgical procedures given in a lucid practical style.

There are some twenty sections in this volume and the illustrations in each section start from 1 onwards; this in itself is somewhat confusing and does not make for comfort or efficiency. We hope in future editions this will be remedied.

The editor has managed to weld into one volume the articles of thirty-four surgeons and he has done his work well.

BOOK NOTICES

[*The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest for our readers.*]

The 1941 Year Book of Industrial and Orthopædic Surgery. Edited by CHARLES F. PAINTER, M.D., Orthopædic Surgeon to the Massachusetts Women's Hospital and Beth Israel Hospital, Boston. $7 \times 4\frac{5}{8}$ in. Pp. 432, with 353 illustrations. 1942. Chicago: The Year Book Publishers, Inc. $3.00.

Anæsthetics Afloat. By Surgeon Lieutenant-Commander RONALD WOOLMER, R.N.V.R., B.A., B.M., B.Ch. (Oxon.), D.A., Anæsthetic Specialist, R.N. Medical Service; Late Senior Resident Anæsthetist, St. Thomas's Hospital, London, etc. With an Introduction by Surgeon Captain H. D. DRENNAN, D.S.O., R.N. $7\frac{3}{8} \times 4\frac{7}{8}$ in. Pp. 120 + x, with 18 illustrations. 1942. London: H. K. Lewis & Co. Ltd. 6s. net.

Acute Injuries of the Head. Their Diagnosis, Treatment, Complications, and Sequels. By G. F. ROWBOTHAM, B.Sc. (Manch.), F.R.C.S. (Eng.), Late Hunterian Professor of Surgery and Dickinson Scholar; Surgeon in Charge, Department of Neurological Surgery, Newcastle General Hospital; Neurological Surgeon, Royal Victoria Infirmary, Newcastle-on-Tyne; etc. With a Foreword by NORMAN M. DOTT, M.B., Ch.B. (Ed.), F.R.C.S. (Ed.). $9\frac{3}{4} \times 6\frac{3}{4}$ in. Pp. 288 + xii, with 124 illustrations. 1942. Edinburgh: E. & S. Livingstone. 25s. net.

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